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A REVIEW OF TUBERCULOSIS IN THE FIELD OF OTOLARYNGOLOGY.

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Myerson[1] reviews the history of tuberculosis, and particularly laryngeal. He very properly called attention to the advances made in diagnosis and treatment following the discovery of the laryngoscope by Garcia. Prior to 1855, syphilis, carcinoma and tuberculosis of the larynx were confused. He mentions the lowered incidence of both pulmonary and laryngeal tuberculosis with improved care of tuberculous patients all over the United States. He believes patients with pulmonary tuberculosis should have a laryngeal examination at least once a month, and that infection of the larynx takes place oftener through the blood stream than by contact with sputum. The incidence of laryngeal tuberculosis in males and females; the ages at which the disease is most prevalent; the importance of occupation; previous disease of the nose and throat; acute and chronic laryngitis; lues; and the general health are all discussed. He quotes from 32 articles in the literature.

Myerson[2] in this article presents the pathology of laryngeal tuberculosis. He emphasizes the many and variable lesions produced by the disease in the larynx. He reviews the anatomy, followed by historical data. He classifies the lesions as infiltrative, proliferative and destructive, with subheads under each one. His Table I shows an analysis of solitary or isolated lesions, the distribution of lesions, the number of times each lesion is found in each part of the larynx, etc. He presents photographs of gross and microscopic specimens to illustrate

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the lesions. Each and every detail of the pathology is discussed at length.

Myerson[3] presents the many and variable symptoms of laryngeal tuberculosis. He discusses the lesions causing hoarseness, such as those on the vocal cords, ventricular bands, in the ventricle, in the arytenoid space, at the anterior commissure, in the arytenoids and in the cricoid. He says pain may be spontaneous, or unprovoked, and provoked. It may be local or referred. He discusses the disturbances of swallowing, pain without hoarseness, dyspnea, cough, dryness, exudate, etc. Under diagnosis he emphasizes the importance of the differential diagnosis of syphilis, carcinoma, lupus and leprosy. He supplements the diagnosis by the Roentgen-ray diagnosis and direct laryngoscopy. Under prognosis he considers complications, an analysis of the sputum findings, the benefit to be derived from pneumothorax, type of lesion in the larynx, pregnancy, etc. He concludes the article with seven case reports.

Myerson[4] mentions the difficulty of finding a satisfactory treatment because there are so many different types of laryngeal lesions and we haven't a specific remedy. He says treatment is carried out for two main reasons: symptomatic palliation and the healing of the lesions. Active disease must be managed and treated differently than inactive. The general condition of the patient, the extent of the pulmonary and the laryngeal tuberculosis must all be considered. He mentions cocaine sprays, narcotics for sedation and the cautery for painful ulcerations. He lists a long series of remedies which from time to time have been recommended. He enumerates the important remedies, such as the cautery, vocal rest, oils, light therapy, X-ray therapy, injection and resection of nerves. He discusses at length the value of each and the method of use. In conclusion, he analyzes the 145 cases healed from 1,000 cases. Fibrosis must take place in the larynx for healing. If Nature doesn't produce fibrosis, no amount of local treatment will cure the larynx.

Hersh[5] reports a case of tuberculosis of the maxillary sinus in a Porto Rican woman, age 26 years. Following pneumonia, she had pain in the right cheek. A radical operation was performed for chronic suppurative right maxillary sinusitis.

Following the operation there was a fistulous tract extending from the right antrum to the gingivolabial fold. For several months before admission to the hospital she had abdominal pain. A gradually enlarging mass was found in the right groin. This was aspirated twice and the fluid contained tubercle bacilli. These were demonstrated by culture and by animal inoculation. Roentgen-ray studies showed destruction of the lower border of the second lumbar vertebral body and the upper border of the third. The antrum filled with polypi in spite of the irrigations. Biopsy showed nonspecific granulation tissue and polypi. After six months the orbit became involved on the right, and the right external rectus muscle was weak. The right frontal sinus was also cloudy. At an exploratory operation necrotic bone was found at the bottom of the floor and lateral aspects of the right orbit. Vision diminished and there was more destruction of the orbital floor with involvement of the right ethmoid and frontal sinus. Occasionally large sequestra were removed. On account of the patient's poor condition, more radical surgery was deferred. He reviews the literature consisting of 19 articles. He quotes Gleitsman, who has reported 20 cases of tuberculosis of the maxillary sinus. Most of the patients showed an extension to the maxillary sinus from a nearby focus in the nose or superior maxilla. Most of the patients had pulmonary tuberculosis. Hersh found the prognosis poor, as only six of the 26 reported cases in the literature were cured. He believes the cure is mostly likely with an early diagnosis and radical surgical intervention.

Spira[6] gives great weight to the importance of the hematogenous origin of laryngeal tuberculosis, especially when the physical examination of the chest and the Roentgen-ray films are negative. Even when the lungs show some involvement, he believes the hematogenous origin is important. He classifies two clinical forms of laryngeal tuberculosis, as follows:

1. A malignant type which begins with intensive symptoms at the entrance to the larynx and spreads rapidly into the larynx proper. This swelling of the rim of the larynx can be easily seen with the mirror. *2.* The type which usually begins as an infiltration about the vocal cords and which later becomes ulcerated. The second form is more benign and may heal spontaneously after lasting some time.

Morlock and Hudson[7] believe bronchoscopy helpful and that it is indicated in five types of pulmonary tuberculosis, as follows: *1.* Cases with negative sputum. *2.* Patients showing areas of collapse. *3.* Cases with areas of obstructive emphysema. *4.* Patients with symptoms of tracheobronchial tuberculosis. *5.* Cases in which some other disease is present with tuberculosis. They present some illustrative cases in which the signs and symptoms are present.

Kleinfeld and Smith[8] report a Porto Rican female, age 3 years, who complained of diarrhea and loss of weight when admitted to the hospital. One year before, she had earache, and two months before admission the right ear had discharged. The mantoux test was negative. The ova of schistosoma mansoni were found in the stools. Tartar emetic intravenously made them disappear. The evening temperature was 102°. X-rays of the chest showed infiltration of the right hilus.

One month after admission, the right side of the face was paralyzed. There was cheesy material in the right auditory canal. There was a large perforation of the drum. The X-rays showed a dense mastoid. A radical mastoidectomy was done. There was slight destruction about the antrum. Culture showed the streptococcus viridans. The facial paralysis disappeared in two weeks, but the fever and diarrhea persisted. A month later the facial paralysis reappeared. Six weeks later, there was marked fluctuation over the mastoid scar. This was reopened. Smears and cultures were negative for tuberculosis. Five days later, necrotic bone was found in the mastoid. The spinal fluid was negative. The child died after eight months in the hospital.

The autopsy showed miliary and pulmonary tuberculosis with old infection of the intestines and liver by schistosoma. Tuberculomata had eroded the facial canal. The authors believe the tuberculomata invaded the petrous pyramid.

Ormerod[9] reports 10 cases. He used for treatment lactic acid, guaiacol and trichloracetic acid. He found high frequency cauterization best of all of these for the mouth and pharynx. He likes the actual cautery better for laryngeal tuberculosis. He uses a needle electrode for the edge of the ulcer, and a ball electrode on the base under local anesthesia with cocaine.

Lapine[10] recommends the use of flexible, radio-opaque, two-way flow catheters of Hicquet. These are about 45 cm. long and of 6 mm. calibre. The ends are pointed. He uses 2-4 cc. of 2 per cent cocaine in cherry laurel water. This has phenol and adrenalin added and this is dropped through the nose with the head thrown back. This solution is applied to the trachea. The catheter is introduced through the nose into the right and then the left main bronchus. The fluoroscope is used to guide this. Ten cc. of normal saline or distilled water is instilled. After several minutes, 2 to 5 cc. are aspirated into a sterile tube. By repeating this on the other side, separate specimens are provided for the laboratory.

Urbantschitsch[11] examined and treated four cases of lupus of the ear, from 1907 to 1913, involving the lobule. He employs surgery and Finsen irradiation. He mentions the disappearance of lupus of the lobule. He attributes this to earlier diagnosis and the nonuse of ear rings. He also observed quite a difference between the subjective and objective course of histologically proved tuberculosis of the matsoid. When there was marked destruction of the mastoid, the general condition of the patient was favorable. He found tuberculosis of the middle ear in two infants and one old woman, age 70 years. He believes these were clinically primary. He found the prognosis favorable. He also reported three cases of miliary tuberculosis evidently precipitated by surgery. All three ended fatally. The operations were very necessary in an effort to save human life. He describes nontuberculous mastoiditis in a patient with tuberculosis. One family history led him to believe tuberculosis may be the basis for the development of deafmutism.

Tardy[12] quotes Pochitonov, who observed a series of 5,000 children in a hospital for the care of pulmonary diseases. Only 2,060 of these had tuberculosis. These were as follows: Chronic tuberculous intoxication, 38.8 per cent; tuberculosis of bronchial lymph glands, 31 per cent; pulmonary tuberculosis, 21.1 per cent; tuberculosis of the cervical lymph nodes, 6.2 per cent; tuberculous pleuritis, 2.25 per cent; tuberculous meningitis, 0.25 per cent; peritonitis, 0.25 per cent; lupus, 0.1 per cent; and tuberculosis of the larynx, 0.05 per cent (one case). Frischman, also quoted by Tardy, reports on the pathological specimens from St. Olga's Children's Hospital from 1887 to 1921. His report is as follows: Spleen involved in 86 per cent of the cases, the liver in 73 per cent, the lungs in

57 per cent, the meninges in 56 per cent, the kidneys in 46 per cent, the intestines in 24 per cent, the pleura in 16 per cent, the brain in 10 per cent, the mesenteric glands in 6 per cent, and the peritoneum in 5 per cent. The larynx is not given. The author regards the ductless glands and the heart as immune. Pochitonov's report was from Leningrad, Russia, and Frischman's from Moscow, Russia.

Tardy quotes Teyschel, of Vienna, Austria, as follows: The figures on the positive reaction to tuberculin in children was as follows: From 1 to 2 years old, 9 per cent; from 3 to 4 years old, 27 per cent; from 5 to 6 years old, 51 per cent; from 7 to 10 years old, 71 per cent; and from 11 to 14 years, 94 per cent. He found eight cases of laryngeal tuberculosis in children from 10 to 15 years old in the division of the hospital for children during the years 1921 to 1934. During this same period there were 986 cases of laryngeal tuberculosis in adults. The percentage in children was 0.8.

Csont[13] reports a boy, age 2 years, who was admitted to the hospital with a diagnosis of spontaneous pneumothorax. At an examination, three months before, tuberculosis at the hilus was discovered. There was very limited breathing on the left and the left side of the chest was enlarged. The mediastinum was displaced to the right on the X-ray film. The diaphragm was displaced down on the left. On the left, in the hilus, there were two lymph nodes about 2 cm. in diameter. These were not present three months before. The emphysema was caused by partial compression of the left main bronchus. X-rays made three months later showed a normal lung and the lymph nodes much smaller.

Colledge[14] reports a case of tuberculous stenosis of the trachea. The patient had apparently recovered from tuberculosis of the lungs, larynx and pharynx. He developed severe tuberculosis of the trachea. This was benefited by a tracheotomy. The patient gained in weight, his voice was clear, he slept better and was more comfortable for months. Death came suddenly, apparently from a cardiac crisis. The cough was exhausting and there was irritation from the tube. An autopsy was not performed.

Alonso[15] calls attention to the timely fact that a tuberculous patient may have many diseases of the larynx, just as the

nontuberculous do. Lesions isolated in the larynx and those existing with tuberculosis are mentioned. There may be many difficulties in making the differential diagnosis. He mentions the following: Catarrhal laryngitis may be due to the constant irritation from coughing. There may be dysphagia. Voice rest helps. Monocorditis, laryngeal pachyderma, nodular corditis, contact ulcer, aphthous ulcers, diphtheria, paresthesia, spasm disorders of motility, etc.

Roux[16] reports a patient who suffered from a generalized tuberculosis, apparently not miliary. There was a tuberculous disease of the left mastoid. Adequate mastoid surgery failed to improve the diseased mastoid. A massive hemorrhage from the internal carotid produced a fatal termination.

Terracol and de LaGrange[17] believe the rarity of auricular tuberculosis is a clinical illusion, as quoted from Lermoyez. Because secondary infection is nearly always present, the clinical picture may have been misleading. The cases may be very mild, moderately severe or very severe. They report four cases. The first was a child, age 3½ years, who died of septicemia following aural surgery. Animal inoculation was positive. The second patient was a man who developed a parotid abscess. Aural polypi were removed. This was followed by a cold sternal abscess. The skin test for tuberculosis was positive. Meningitis threatened, so that mastoid surgery was performed. The carotid canal was involved. There was a fatal hemorrhage one month later. Autopsy showed primary temporal bone tuberculosis with secondary invasion of many other organs of the body. The third was a child, age 2 years, with recurrent pulmonary tuberculosis. Both ears were tuberculous. Direct smears, histologic examination and animal inoculation were all positive. After one year, the child was in good health. The fourth case was a man with bilateral pulmonary tuberculosis and an aural polyp which was microscopically tuberculous. The authors give clinical and anatomical classifications. They urge heliotherapy following surgery.

Alonso[18] believes polypi, cysts and benign tumors of the larynx may not be tuberculous even in patients with pulmonary tuberculosis. Prompt healing may follow removal. He believes earlier diagnosis of syphilis and better treatment has decreased the laryngeal lesions in the tuberculous. He

attributes the cicatrices in the larynx to either lues or radium. He mentions the importance of the differential diagnosis of tuberculosis, lues and carcinoma. He mentions 435 cases of carcinoma of the larynx, in 18 of which tuberculosis was also present.

Warren, Hammond and Tuttle[19] report the results from 198 patients with tuberculosis of the lungs examined bronchoscopically. Of these, 74 had tracheobronchial tuberculosis in the form of ulcers or granulations. Other tuberculous lesions were also found. They found cauterization of the ulcers, removal of the tuberculomata and dilatation of the stenosed bronchi helped the patients. The lesions healed in 34 of 57 cases treated. In 10 there was no improvement. In nine others it was too soon to learn the results of treatment.

Davison[20] treated eight patients with an electrocoagulation current of weak intensity. The lesions in the trachea and bronchi had been present for some time and had shown little tendency to heal. His results were uniformly good. He advocates an interval of two to four weeks between the bronchoscopic treatments.

Schneider[21] finds the Roentgen-ray and bronchoscopic examination necessary for the diagnosis of stenosis caused by enlarged mediastinal lymph nodes. The cause of the stenosis was subsequently proved. At first, bronchial asthma or a foreign body seemed more likely. The stenosis was due to compression of the right main bronchus.

Myerson[22] believes the bronchoscope is of more importance for diagnosis than for the treatment of tracheobronchial tuberculosis. He finds the cause of the atelectasis may be a tuberculoma or fibrotic lesions. With the exception of coagulation and cauterization of tuberculomata and ulcers, he could do little of positive value. He condemns solutions of silver nitrate stronger than 10 per cent. He found it very difficult to dilate the cicatricial stenoses, and even dangerous. He believes recovery depends upon Nature's ability to heal the process as very frequently bronchoscopies are not practical.

Myerson[23] reviews 1,000 cases of laryngeal tuberculosis and presents many of the features of the disease given in more detail in his four previous articles. He shows 12 tables. His article covers pathology, both gross and microscopic, with

photographs, the different types of clinical lesions, the symptoms, diagnosis, prognosis and treatment.

Rubin and Galburt[24] mention the infrequent appearance of both pulmonary and laryngeal tuberculosis in children. The authors reviewed the literature of the laryngeal disease in children, beginning with 1806. Of 115 children in the pediatric service at Sea View Hospital, 49 had laryngeal changes. Of these 49, 30 had tubercle bacilli in the sputum, feces or gastric contents. The authors mention the methods of infection and list them in their order of frequency, as follows: *1.* By way of the lymphatics from the primary pulmonary lesion. *2.* By direct contact with the bacilli-laden sputum. *3.* Hematogenous infection. *4.* By direct extension from a pharyngeal lesion.

They found the laryngeal symptoms milder than in the adult. All the children were examined by the indirect method. The most frequent lesion was hyperemia. Infiltration was occasionally found. Granulations, edema, ulceration and perichondritis were rarer. The children were between the ages of 3 and 15 years. Forty-nine per cent showed some laryngeal involvement. Treatment consisted of instillations of halibut liver oil or cod liver oil, ultraviolet irradiation, the galvanocautery, injection of the superior laryngeal nerve for pain, vocal rest, a spray of 1 per cent solution of cocaine, etc.

Bordley and Baylor[25] report 79 patients examined and observed between the years 1912 and 1924. At operation, the tonsils and adenoids were found to be tuberculous. The follow-up was made in 1935. They were able to hear from 45 of 47 patients for whom operations were performed before they were age 14 years. One of these had died of tuberculous meningitis, and two had died of a nontuberculous disease. All of the balance of 45 were living and well. Two patients who could not be traced were well some months after operation. Not any of the patients had acquired pulmonary tuberculosis. Many of the patients had a tuberculous cervical adenitis at the time of operation. There was no evidence of a progressive pulmonary tuberculosis.

Hallberg[26] reports one case from the Mayo Clinic, and mentions Havens' five cases previously reported from the same clinic. He calls attention to the infrequent occurrence of the

disease, due to the greater resistance of the nose to tuberculous infection. The lesion is usually a granuloma on the septum or one of the lower turbinates. Ulceration may occur later. Scarring and stenosis of the meatus may follow healing from fibrosis. The disease must be differentiated from syphilis, lupus and malignant growths.

Alpert[27] presents a table showing data of 21 cases in which tuberculosis was found in surgically removed tonsils. His table shows what other organs of the body were also tuberculous. He has reviewed the work of others who have reported their findings. Of 202 tonsils removed, 21, or 10.4 per cent, showed tuberculous infection. None of the tonsils gave clinical evidence of tuberculosis before operation.

Lukens[28] emphasizes the importance of early diagnosis and treatment of pulmonary tuberculosis in order to prevent laryngeal invasion. He refers to Minor's work in examining the larynx frequently in patients with pulmonary tuberculosis to detect and treat the laryngeal disease early. The early lesions rarely produce symptoms. He discusses some of the more frequent laryngeal lesions and their location.

Long, Seibert and Gonzales[29] report the decline of tuberculosis of the tonsils, which follows the decline of the disease in other organs. They refer to the reports of Magee and Weller and call attention to the high death rate from tuberculosis among the American Indians. The authors secured 1,000 pairs of tonsils removed from Indian children and young adults. These were from many different Indian tribes, but more than one-third were from the Navajos. By way of comparison, 600 pairs of tonsils were secured from Porto Rico, and 400 pairs from Philadelphia. They found tuberculosis in 6.5 per cent of the tonsils from the Indian reservations; 2.5 per cent from Porto Rico, and 0.3 per cent from Philadelphia.

Woodward[30] calls attention to the marked advancement of thoracic surgery as applied to pulmonary tuberculosis, with better prevention and treatment of the larynx. He reviews 136 cases treated during a five-year period of 1923 to 1928, as compared with 152 cases treated during a five-year period from 1933 to 1938. Both groups had local galvanocautery treatment of the larynx, but the first group had almost no collapse therapy except for 4.4 per cent. The second group

had collapse therapy applied to 49.3 per cent. His report is based on 288 patients with laryngeal tuberculosis out of 2,129 patients admitted to the sanatorium. On admission 12.8 per cent had laryngeal tuberculosis. In the second group, 50 per cent showed improvement.

Cotton-Cornwall[31] considers pain the worst symptom of laryngeal tuberculosis. He mentions the different standard methods of relieving the pain, such as Wolfenden's position, aspergum, anesthone, euphagin, anesthetic powders and ionization by electrical anesthesia. He uses the current from a 45-volt dry battery controlled by a reducing coil. He describes the electrodes and the method of using them. Lint wet with 2 per cent potassium iodide is used about the negative pole, and water about the positive pole. The negative pole is placed over the larynx, and the positive over the nape of the neck. He uses 10-12 ma. He begins with 4-5 ma. Above 9 ma., the skin may be burned. Applications last one-half hour and are repeated each day until the anesthesia lasts 24 hours. After this the treatments are repeated as needed. Usually six or seven applications are sufficient to begin with. He quotes Grain's work in 1936. A brief summary of 11 cases is presented.

Pollard and Combs[32] conducted a study of 107 patients following the removal of tuberculous tonsils. They give due credit to others who have reported their findings. Ninety of the 107 patients had a chest Roentgen-ray film made, either just before or just after operation. Sixty-three were followed for an average time of 4.4 years. Fifteen had pulmonary tuberculosis. Six had tuberculosis of the mediastinal lymph nodes. Twelve had tuberculous involvement of the cervical lymph nodes. Eight had tuberculosis of the bones and joints. Seventy did not have any discoverable tuberculous lesion at the time of operation. They give brief reports of 36 of their cases. Their report shows the decreasing incidence of tuberculosis of the tonsils.

Derscheid and Toussaint[33] have called attention to the old belief that the lungs were the only site of tuberculosis, and a fatal outcome seemed inevitable whenever metastatic lesions appeared, such as those of the intestine or larynx. Modern progress has shown the frequency as well as the curability of many of the localizations which complicate pulmonary tuber-

culosis, of which tracheal tuberculosis is one. The syndrome of tracheal tuberculosis, which is sometimes noted in pulmonary tuberculosis, includes sizable exudative lesions occupying the lower third of the glandular system of the trachea. It is marked by fitful, irritating cough which provokes vomiting at times, and is either dry or followed by foamy, white expectoration. The authors call this syndrome — which evolves independently from the contingent pulmonary foci — tracheal, and not tracheolaryngeal or tracheobronchial, in order to distinguish it from laryngeal tuberculosis, with or without tracheal invasion, and from bronchial tuberculosis. No visible or lasting laryngeal lesions exist in tracheal tuberculosis, but frequently there are edema and congestion. The cough is fitful, loud, and appears regularly in after-dinner or evening crises. Laryngeally, hoarseness — and sometimes aphonia — and redness of the cords are evident. The authors describe a method of rapid laryngeal anesthesia which was devised by them to alleviate these coughing crises, and they remark that the syndrome in question is observed in various forms of pulmonary tuberculosis. Postmortem findings are then discussed, and the following conclusions reached: Specific lesions of the submucosal lymphatic system are constant, and in the same patients the tuberculous lesions of the glandular system of the trachea are clinically unimportant as long as they do not spread to the two upper thirds of the trachea; when they do spread there, the tracheal syndrome appears. Tuberculous formations of the glands in the lower third of the trachea, and lymphatic lesions of the entire submucosa, are as usual as bronchial lesions in patients with pulmonary tuberculosis. There is an extensive general discussion and a review of the literature, but no bibliography.

Bernfeld[34] discusses several clinical features of tuberculosis of the larynx which are usually neglected, with the hope that his paper will stimulate further research. He discusses the etiology of the disease and states that differentiation of hematogenic from contact infection in the presence of active pulmonary tuberculosis is difficult, especially in advanced cases. A negative sputum is practically always proof of hematogenic infection. Hematogenic infection in the presence of trivial pulmonary findings is demonstrated by two personal cases. The epithelium of the larynx is relatively resistant to contact infection. Experiments with guinea pigs have shown

that the inhalation of 50 tuberculosis bacilli invariably causes pulmonary but never laryngeal tuberculosis. Cultivation of bacilli from blood has been found difficult. Clinical manifestations help in the diagnosis of hematogenic infection. The characteristic symptoms are eruptions of the mucosa, in rapid succession or simultaneously; to some extent, manifestations of several foci in the body. One personal case is related.

Predisposition: Continuous coughing eventually causes lesions of the laryngeal epithelium, which facilitate deposition of sputum containing bacilli. The author contradicts Tövölgy's theory, based on extensive investigations, that coughing is a protective reaction; he concedes, however, that moderate coughing may have a protective effect, dependent on the character of the sputum, time of incidence, etc. In the presence of severe coughing in pulmonary tuberculosis, the larynx is involved in 24 per cent, in the absence of coughing in 6 per cent of the cases. Laryngitis is undoubtedly an important factor in pulmonary tuberculosis and creates a soil eminently favorable for infection. The author refers also to increased acidity of the inflamed vocal cords as a predisposing factor (Schade). One personal case is presented to demonstrate the susceptibility of chronic unspecific cases to being transformed into specific lesions. Recurrent laryngitis associated with pulmonary tuberculosis is a predisposing factor as well. Vocal disorders have symptomatic significance. Stagnation of phlegm has an influence on the prognosis as well as the therapy of a case, for two reasons: it may be the result of deficient expulsion in normal throats, and it may be caused by qualitative and quantitative modifications of secretion. In rare instances stagnation is recognized by impaired mobility of the epiglottis. Two cases are described.

The author believes that altered secretory function in tuberculosis is responsible for troublesome dryness of the upper respiratory tract in tuberculous patients, especially during the spring and fall, when the hot desert winds blow (Palestine). Tuberculous ulcerations of the larynx heal spontaneously (clinical healing) in some cases, especially, if not exclusively, in hematogenic infection. The tonsils as factors: Many conditions may simulate tonsillar disease. The altered secretory function in tuberculosis should always be borne in mind before tonsillectomy is performed with that disease. Hypersecretion

is no indication for tonsillectomy, even in manifest tuberculosis of the tonsils, and operation will be useless. Infection of the larynx from the tonsils is possible only by way of the deep cervical glands, as there exists no direct lymphatic connection between the larynx and the tonsils (Safranek). Conversely, however, the tonsils may become infected from either the lungs or the larynx by sputum thrown upon them by coughing; hematogenic infection from these sources is also possible.

Rocca[35] reviews the growing practice of laryngologists in the treatment of pulmonary tuberculosis, and the statistics on the frequency of tuberculosis of the larynx. The author considers the latter's proportion between the sexes, noting that although their ratios vary between three men to one woman and two men to one woman, most authors report a greater incidence of the disease in men, as he himself found in one survey (3,492 patients, 114 cases of acute laryngeal tuberculosis in men, 44 in women). He discusses the difficulty of attaining correct statistics owing to borderline cases, contributory factors and other complications, and rejects the practice of calling doubtful cases "pretuberculous laryngitis," observing that they either are tuberculous or are not. The results of the examinations of 740 women patients with pulmonary tuberculosis appear in a table which presents the incidence of the various forms of laryngeal tuberculosis. Another table records postmortem findings of 133 women patients. The proportions between the various forms of pulmonary tuberculosis and laryngeal tuberculosis follow, the author concluding, in round figures, that there is one patient with evident laryngeal localization in every five women patients with pulmonary tuberculosis. There is a general discussion but no bibliography.

Dierichs[36] reviewed Grain's method of electrical anesthesia and used it. His results are pronounced satisfactory. Certain shortcomings of the method will be overcome with greater experience. The treatment affords, of course, only symptomatic relief, but it has a great psychological effect on the patient. It enables patients to eat, and in the end makes their death light.

Norrié, Gomes Veiga and Lozano[37] believe a careful examination of the larynx in patients with pulmonary tuberculosis

is advisable, because the latter is not infrequently associated with tuberculosis and carcinoma of the larynx. The authors point with special emphasis to the immobility of a vocal cord as a very significant symptom of a possible carcinoma. In tuberculosis of the larynx, immobilization of a vocal cord is met with only in rare instances. A case is presented. A patient with tuberculosis of the lungs and larynx, whose vocal cord was fixed. An exploratory excision showed tuberculosis of the larynx and carcinoma.

Rosenthal[38] believes the diagnosis and therapy of tuberculous laryngitis needs improvement, beginning with a revision of the ideas of phthisiologists, for most of whom the respiratory system begins at the trachea. It is an easily diagnosed disease, since with rare exceptions it is secondary to pulmonary tuberculosis, and is recognized at onset by vocal disturbances. Diagnostic errors result at times, however, when imperfect coaptation of the cords is mistaken for laryngitis and tuberculous infiltrations of the lungs. Although infection may be carried by the lymphatic system and the blood stream, bacilli in the sputum are the principal cause of laryngeal tuberculosis. Thus, efforts should be made to purify the sputum. General treatment is beneficial but the author considers local therapy most important, with excellent results. He has reviewed the literature and has six bibliographic references.

Spira[39] says tuberculosis of the larynx is practically never a primary disease. It usually occurs in the stage of generalization of tuberculosis or the stage of localization in the lung. Contact infection is an obsolete conception. Tuberculosis of the larynx is observed with and without the presence of coughing. The author has never been able to note any parallelism between catarrh and the incidence of tuberculosis of the larynx. On the contrary, he has always had the impression the lesions occurred most often in patients whose larynges had previously been sound. Causation is mostly metastatic or hematogenic. Bacillemia has been reported in the literature in varying degrees. Tuberculosis of the larynx in the presence of hematogenic pulmonary tuberculosis is most probably due to hematogenic infection. Not infrequently the examination is negative for pulmonary tuberculosis, but there may be extrapulmonary foci. The author tabulates the cases that have come under his observation (112) and arrives at the

conclusion there exists a certain relationship between the laryngeal lesions and the manner of dissemination of the pulmonary process. In the bronchogenic form of pulmonary tuberculosis the interior of the larynx is the locality most often involved. In the hematogenic form the lesion is more often localized in the external ring, but in this case the interior also becomes involved. The value of these findings, according to the author, is relative because the findings represent the conditions only during a single month and cannot take into consideration any previous and subsequent changes in a process over periods of years. The author points out that his findings with regard to hematogenous tuberculosis of the larynx are in direct opposition to the findings of most investigators. There are clinical descriptions and several histories. The author notes the disease became manifest in some of his patients following severe physical exertions.

Knapp[40] reviews the question of hematogenic infection of the larynx on the basis of the histological findings in 20 fresh tuberculous larynges and a series of older preparations, and contributions are made to several other questions of pathological anatomy of tuberculosis of the larynx. On the strength of histological findings viewed in the light of the total autopsy reports, hematogenic infection appeared absolutely certain in three cases, and highly probable in two other cases. These instances cannot serve as proof for the theory of primary hematogenic infection, because tuberculosis of the mucosa existed in all five cases; they merely indicate that hematogenic implantation may occur in the larynx also without the presence of miliary tuberculosis. The only case of primary tuberculosis of the larynx acceptable among all the cases reported in the recent literature is the one described by Richter in 1929. Involvement of the regional lymphatics is rare because tuberculosis of the larynx is as a rule a secondary infection. A defensive reaction of the epithelium against the invading tuberculous granulation tissue is noted only in the pavement epithelium. Proliferative processes are not visible in cylindric epithelium. An eminent power of resistance to the destructive incursions of tuberculosis is evidently possessed by the secretory ducts of the mucous glands. The mucous glands themselves, on the other hand, are sites of predilection of the tuberculous process. The occasional presence of solitary tubercles in the circumscribed, large-meshed edemata argue for the

inflammatory nature of the edema. The filiform cells which Hosomi described in tuberculomata of the cartilaginous nasal septum are apparently absent in the cartilage of the larynx. On the other hand, however, a calcareous infiltration like the one described in a tuberculous larynx by Schottelius was found in the region between the cartilage and the ossified part of the organ.

Indifferent or disastrous past results have induced Predescu-Rion[41] to seek a reliable test of biologic reactions that would determine the use or omission of the galvanocautery. He found it in a modification of the Westergren sedimentation test, as practiced by Fath. The rate of hourly sedimentation, expressed in millimetres, supplies in this test a differential (Arnold's) between the readings of the first and third hours, and since repeated sedimentation tests are practiced in each case, several differentials are available. The relation between the differentials represents the patient's biologic stability, or his lack of it, a low differential ordinarily indicating recovery, irrespective of the first-hour reading. This does not apply, however, when the first-hour figure of a test surpasses 30 mm. The author offers the case history of a patient treated before adopting the sedimentation test, and five case histories of patients treated with its assistance. The author believes the results obtained (one recovery, four deaths) confirm the conclusions of Fath and Arnold, and the sedimentation test indicates correctly the biologic deficiency of the patients in the fatal cases, and the favorable biologic reaction in the ameliorated one. There is a general discussion and a review of the literature, but no biblography.

Denoyer[42] reviews the forms of tuberculosis of the oropharynx, affirming that primary infection is possible in that organ. Studying the relations existing between tuberculosis of the tonsils, and of the larynx and lungs, he demonstrates that while the ratio of oropharyngeal tuberculosis to pulmonary is only 2 per cent, that of the laryngeal form to the latter is 20 per cent. Studying tonsillitis and its modes of inception and diffusion, he divides pharyngotonsillary tuberculosis into the rare primary form, a case history of which is given, and the more frequent secondary, presented here with two histories. A case history of chronic ulcerous tuberculosis of the tonsils follows, after which the author presents one of the

much rarer lupus forms, and one of latent pharyngeal tuberculosis. The therapy of the forms is then discussed, their etiology and histology, and the author's own theory about the disproportion between the ratios oropharyngeal-to-pulmonary and laryngeal-to-pulmonary case percentages, the theory being based upon the stronger antibacterial defenses of the oropharynx. He has a bibliography of 23 titles.

Collet[43] believes if tuberculous tumors of the larynx are infrequent, subglottic ones are rare. They represent a localization of the former and are sometimes mistaken for tracheal tuberculosis. The author presents a case history: A man, age 36 years, who had had pulmonary tuberculosis and artificial pneumothorax for 16 months, with hoarseness and dyspnea for the same length of time. There were infiltrations of the right lung and lesions of the left apex. Laryngoscopy showed a subglottic tumor; tracheotomy was indicated and performed; thyrotomy was deferred 14 days owing to fever. Two round, hard, smooth, pea-sized tumors with a common peduncle, implanted anteriorly and below the glottis, were excised. The trachea promptly closed, the cannula being removed in 11 days. Microscopically, the tumors showed a quantity of giant cells, whose characteristic aspect differed greatly from that of common laryngeal tuberculosis, in whose ulcerated mucosa there were large, discolored spots in processes of hyaline degeneration or caseation, with a few giant cells. Another case history is presented, which also appears in the author's "Traité de la Tuberculose du Larynx." This book contains a bibliography of the cases of tuberculous tumors of the larynx since 1913. There is a general discussion, but no bibliography.

Effenberger[44] presents a review of the present status of tuberculous disease of the larynx from the standpoint of general treatment. The article is interesting because of the accompanying 11 photographic illustrations of the various tuberculous conditions of the larynx. The pictures were taken with a special camera developed at the author's institution.

Alonso[45] discusses polyps, cysts and benign tumors in the tuberculous larynx, and syphilitic lesions in the nontuberculous and tuberculous larynx. Malignant tumors are then considered, with the presentation of 18 tuberculous cases selected from 435 cases of epithelioma of the larynx. The author believes laryngeal neoplasms in tuberculous patients to be less

rare than is generally realized, adding that modern radiology reveals lesions which could not be discovered by older methods. Surgery is usually better tolerated than Roentgen-ray treatment. Adenopathies in patients with pulmonary tuberculosis are not less frequent than in nontuberculous patients with cancer of the larynx; exceptions are the cases in which laryngeal tuberculosis and cancer coexist. Gangliar currettage produced no evidence of tuberculous lesions of the ganglia in any of the cases in which the larynx exhibited purely epithelial lesions. The evolution of laryngeal neoplasms was not slower in tuberculous than in nontuberculous patients. As in most cases observed by the author, the two lesions — cancerous and tuberculous — occupied the same part, or two closely contiguous parts, of the larynx. The author concludes that their coincidence, and even the graft of one upon the other, is possible. There are clear instances of tuberculous lesions — even circumscribed — constituting the seat of an epithelioma. He adds that the coexistence of cancer and tuberculosis in two quite distant and independent parts of the larynx is possible, but this has never been observed by him. There is a review of the literature, with 11 photomicrographs. The bibliography has nine titles on polyps, cysts and benign tumors; 29 titles on syphilis, and 27 on malignant tumors.

Blomroos[46] believes, even if laryngeal tuberculosis has been the source of extensive and thorough studies, there are still several presumptions which are not fully confirmed and many problems which are not completely solved. Among these are the following: *a*. The comparison between the clinical and the pathoanatomical picture of laryngeal tuberculosis. *b*. The ulcerative processes and their origin. *c*. The classification of laryngeal tuberculosis. *d*. The comparison between the different types of pulmonary and laryngeal tuberculosis. *e*. The way in which tuberculosis spreads to the larynx. *f*. The main lines of therapeutic measures for the different types of laryngeal tuberculosis. *g*. Secondary phenomena in laryngeal tuberculosis. The material was, therefore, studied, with reference to these problems, and the following conclusions drawn: The picture of the spread of laryngeal tuberculosis obtained by indirect mirror-examination forms only a small part of its actual extent, and this is particularly true as regards cases of a destructive nature, in which processes that deeply penetrate the tissues may always be expected. The significance of this

fact for active surgical treatment of laryngeal tuberculosis is quite obvious, considering what an infinitesimal part of the actual tuberculous processes we are able to treat in this way. The largest ulcerative processes are always to be found in those parts of the larynx most exposed to mechanical lesions; consequently, now more than hitherto, attention should be paid, *e.g.*, to such mechanical irritation as is caused by coughing and talking. The origin of tuberculous ulcerations is not uniform, and at least the following methods of formation must be considered possible: *1.* By lesions due to mechanical irritants; *2.* by decomposition of the subepithelial tubercle, whereby the epithelium is involved in the necrosis; *3.* by the pressure-necrosis to which the epithelium is subjected by the growing epithelial tubercle; *4.* by the toxicoinflammatory effect on the epithelium of the lymphocytes surrounding the tubercle; *5.* by the secondary infection by tubercle bacilli of epithelial defects evoked by general inflammation. A complete classification of laryngeal tuberculosis must include, besides the purely morphological grouping into infiltration, ulceration, perichondritis and chondritis, tuberculomata and papillomata, information on the productive or exudative nature of the process, and this is of great importance for prognosis as well as therapy. As regards the congruency of laryngeal and pulmonary tuberculosis, it is on the whole fairly complete, at any rate in cases of a more pronounced, either productive or exudative character. Periodical incongruity is due to temporary changes in the local state of immunity, but these changes are scarcely of a persistent nature. As to the theories of the different ways in which tuberculosis may spread to the larynx, an infection via the blood vessels may be considered possible at the generalization stage. That type of laryngeal tuberculosis by which we are most frequently confronted is undoubtedly "the sequential disease of an isolated pulmonary phthisis arising in the tertiary stage," and here contagious infection is the most common. For the therapy, it must always be remembered that the general character of the tuberculosis in question, and the general, as well as the local, state of immunity of the body are of greater importance than the local findings. Active treatment of the local process is, firstly, hopeless in unfavorable cases, considering the real spread of laryngeal tuberculosis, and, secondly, fatal if we consider the inability of the organism to overcome the resulting general as well as local reactions. The glandular

tissues have no predilection for the localization of laryngeal tuberculosis. The changes, both inflammatory and degenerative, occurring in the glandular tissues are of a secondary nature. This is also true of the muscles and the epithelium. The paper is accompanied by 18 detailed case reports with autopsy and microscopical findings, as well as an extensive bibliography.

Caliceti[47] reviewed the complementary function of laryngology in sanatoria in the past, its evolution into periodic but still complementary consultations during recent years, and the inadequacy of both arrangements. He refutes conceptions of laryngeal lesions based wholly upon vocal alterations or dysphagia, showing that serious lesions may exist with slight symptomatology or none, and disproves old statistics on the relative frequency between laryngeal and pulmonary tuberculosis (specific laryngeal lesions in 30 per cent of all cases, and 50 per cent of those in advanced stages), citing his own experiences with submucous tuberculous lesions existing even in macroscopically healthy larynges. After a general discussion, the author reiterates the necessity of establishing a resident laryngologist in every sanatorium, as well as that of a separate ward where patients whose lesions are preponderantly laryngeal may be observed with better care. In concluding, he summarizes his plan thus: all sanatorium patients to be examined by laryngologists; otorhinolaryngologic departments to be established in every sanatorium; a central bureau to be created for the co-ordination and study of the various laryngologic services.

Matushita and Usuku[48] present the therapeutic results of irradiation in 34 patients. Cured six (18 per cent); improvement, 15 (44 per cent); nine unchanged (26 per cent), and four worse (12 per cent). The total of cured and improved cases was 21 (62 per cent). Dosages are given in each group of results and also the laryngeal findings. The X-ray examination of the cured cases disclosed productive and cirrhotic changes in the lungs. In seven of the 15 improved cases, improvement was both objective and subjective. In the remaining eight cases only the objective state was improved. In the former, the lungs presented productive and cirrhotic changes in six cases, and a process of a mixed form in one case. Among the latter, two presented changes of a dissemi-

nated form, two others changes of a mixed form, and the remaining three, changes of a productive form. Among the patients whose condition became worse, the lungs of three presented productive changes, and one exudative changes. Six of the nine patients whose condition was unchanged had prematurely stopped the treatment for personal reasons. The authors arrive at the conclusion that Roentgen-ray therapy is indicated for patients who exhibit neither edema nor ulcers in the larynx. Great care must be exercised in those cases where edema and ulcers are present and the pulmonary process is of a disseminated or exudative form.

Arold[40] presents a tabulation of the therapeutical result secured at his institution. He cites Zange, who observed cures in 71 per cent of patients with productive tuberculosis of the larynx, and only 17 per cent in those with a process of exudative nature. The picture here is distorted because Zange selected his patients from among those whom he believed would be benefited by the treatment. The results of other authors, ranging as high as 60 per cent, are mentioned. The composition of patients at the author's institution is about the same as that met with in other institutions of a like nature. The period reported on covers the years 1933 through 1938. The results, on the negative side (including deaths) fluctuate around 50 per cent and are more constant than those on the positive side, comprising cures and improvements. In the former the range is from 13 to 36 per cent, in the latter from 8 to 18 per cent. As new experience is acquired, the figures on the positive side are bound to go up. According to the diagnosis of the case (mild, medium, severe) the results are as follows: 236 mild and medium cases: improvement, 66.5 per cent; clinical cure 42.2 per cent; 266 severe cases: improvement, 28.2 per cent; clinical cure, 10.9 per cent. In the preceding group, 1930-1932, the percentage of improvements in the former group is higher, but that of cures only half as high as that in the present period. In the latter group both percentages have risen. A separate table presents the relationship between the laryngeal and pulmonary findings. Generally speaking, favorable pulmonary findings go hand in hand with improvement in the laryngeal lesions; in some cases the relationship is parallel. The author further attempts to demonstrate the fallacy that the condition of the larynx is always dependent on the course of the disease in the lungs.

An absolute congruency is manifest only in the results in patients with a favorable pulmonary diagnosis. Improvement is recorded in 14.4 per cent of the patients with decidedly poor pulmonary findings. Most striking in the opinion of the author is the improvement in 87.2 per cent of the patients with dubious pulmonary findings. It is evident from these results that the prognosis in tuberculosis of the larynx is not determined exclusively by a good general immunobiological condition. Rather, a special power of resistance of the organism must also be taken into consideration. A check-up of the patients treated during 1930-1932 showed that 50 out of a total of 157 were still alive at the end of five years. In 48 (96 per cent) of the former there had been no recurrence; the pulmonary processes were arrested in 21, and had advanced in the others. It is further seen that the extent of the original lesion had no influence on the result. Hence, the final results depend not so much on the degree of severity of the original process as on the immediate results of the treatment. Any aggravation of the laryngeal process, with the attendant disorders of respiration and nutrition, is apt to cause a flare-up of every focus in the body of the patient.

After an extensive discussion of other authors' statistical and clinical findings, Raimondi and Nijensohn[50] describe the evolution of one case of laryngeal tuberculosis, and point out in this disease that the symptomatology follows the location and not the extension of the lesions.

The authors stress the importance of systematic laryngeal examinations, which alone will reveal lesions in the zones of the ventricular folds, ventricular ligaments, et cetera; they enumerate the subjective symptoms of the disease (vocal weakness, hoarseness, pain), and add that these were completely lacking in 28 per cent of the cases, and were only slightly evident in the remainder. This paucity of subjective symptoms, and the concordant opinion of phthisiologists and laryngologists about the value of early investigation of this frequent disease prompted the authors to advocate systematic and periodic laryngeal examinations of all patients with pulmonary tuberculosis.

Blomroos[51] discusses, within the space of five pages, the outstanding contributions to the therapy of laryngeal tuberculosis, mostly in the German literature of the past 15 years.

Particular attention is given to irradiation. There is no original contribution.

Weigel[52] believes radioscopy alone cannot determine the presence of pulmonary tuberculosis in the incipient stages. A Roentgenograph has the value of a biopsy in such cases. The evidence of a pulmonary granulation will remove any doubt as to the nature of the infection. The fact that miliary tuberculosis is not radioscopically visible is not sufficiently known to nonradiologists. The author says today it is customary, when treating a patient with primary angina, to test him for diphtheria, Plaut's angina, syphilis, mycosis, lymphogranulomatosis and sarcoma. Only when all that has been done, with negative results, the possibility of tuberculosis is considered, and the patient is sent for a radioscopy of his lungs, to return with a brief note that affirms the absence of any trace of tuberculosis in his lungs. There was no bibliography.

Aloin and Colrat[53] outline the clinical forms of tuberculosis of the eye, nose and their adnexa; their relations, pathogenesis and therapy, as follows: Tuberculosis of the nasal fossae (acute or secondary and chronic; lupus, tuberculoma, osseous tuberculosis) and its complications. Tuberculosis of the eye includes tuberculosis of the lacrimal apparatus and its complications. The diagnosis is easy. The only difficulty is the differential diagnosis from dacryocystitis; in case of doubt, the authors advised inoculations of guinea pigs. Palpebroconjunctival tuberculosis is comparatively rare; conjunctival tuberculosis is more frequent. Both diseases are easily diagnosed, but care must be taken to avoid confusion with Parinaud's conjunctivitis, and a biopsy is sometimes necessary. There are three modes of infection: *1.* By continuity; *2.* by way of the lymphatic system; *3.* by the blood stream. The transmission of the tuberculous infection from the nose to eye is usually by continuity, and occurs much more frequently than from the eye to the nose. In the rare occasions of the latter form, an accidental inoculation of the infection is the probable mode.

A discussion of the relations of the tuberculous nasal fossae to the eye, and of the tuberculous eye to the nasal fossae, as well as of the lesions which are common to both organs, shows how nasal tuberculosis may cause nonspecific lesions by

obstructing the lower orifice of the nasolacrimal duct and specific lesions by extension of the tuberculous process. There is also an inflammatory type of lesion, but it is difficult at times to determine whether this process is nonspecific or specific. Less unusual but still rare are the cases in which the nasal fossae and eye are attacked, either simultaneously or successively. The infection may occur by way of the blood stream. There is a detailed review of pathogenesis and comparisons of the histologic structures of the organs involved, and, following this, therapeutic methods in nasal lesions are outlined. Chemical treatments are not recommended. Surgery remains the only treatment available in lupus, but it has to be repeated, is painful, and its results are only partially satisfactory. Curettage gives better permanent results, but it favors dissemination and may at times cause granular exacerbation, with fatal results. The galvanocautery is inadequate, painful and leaves hard scars. The Finsen light therapy is to be preferred as it leaves only soft, thin scars, but the sittings are long and have to be repeated. High frequency currents, especially diathermocoagulation, are much safer and more efficacious than radium. Fulguration is also used, but its effects are more superficial. Of the therapeutic methods of tuberculous lesions of the eye, catheterism is not recommended owing to the inflammation which may follow. Lavage and injections with mixtures of oil and gomenol or iodoform, iodized glycerine and zinc sulphate are no longer used. Surgery is indicated whenever the lacrimal sac becomes clogged or dilated, requiring excision. It is also recommended in palpebroconjunctival tuberculosis. Tuberculosis of the eyeball is treated medically and surgery should be used only as a last resort. There is an extensive general discussion and a review of the literature. There is a bibliography of 23 titles concerning the nose, and 22 concerning the eye.

A review of the literature on primary tuberculosis of the middle ear is presented by Müller.[54] The author presents his findings in postprimary tuberculosis of the middle ear. Infection is tubal as well as hematogenic, the latter mostly in children. The focus in the ear is in some cases the only focus in the body. There existed also a lymphogenic form of tuberculous otitis media. The hematogenic mode of infection is found in certain forms of cavernous pulmonary tuberculosis. Hematogenic tuberculosis of the middle ear may also start as a

focus in the mucosa or in the bone. A decision whether the infection is hematogenic is often very difficult even with the aid of X-rays. The isolated form is extremely rare. Little is known as yet on the incidence of other forms. Reports of three cases with three Roentgenograms are presented. The relations of tuberculous otitis media to tuberculosis in other parts of the body has a certain diagnostic significance. It is, therefore, advisable to search for other hematogenic foci. Otitis media associated with certain hematogenic metastases, which is not unusual, is suggestive of tuberculous otitis media. Chronic otitis in the presence of pulmonary tuberculosis is very likely of tuberculous origin. It must not be overlooked, however, that pulmonary tuberculosis is sometimes associated with nonspecific otitis media.

Tuberculous manifestations in the external ear are reviewed by Magnoni,[55] who then describes the following case: A woman, age 46 years, familial and personal history negative, ear normal at birth. The lobule was perforated for an earring at 4 years. Two years later, a purplish spot the size of a penny appeared on the lower end of the helix of the left ear, spreading subsequently until it covered most of the lobule at puberty. It continued to grow slowly until the patient sought its removal for esthetic reasons. The patient had a normal constitution. There were negative results from thoracic and otorhinolaryngologic examinations, as well as from Wassermann, Meinicke and Kahn tests. The lobule was enlarged to 32 x 56 mm., nodular and purplish. It was operated and then irradiated. The clinicohistological findings were typical of nodular tuberculosis. There is a bibliography of 31 titles.

Collet and Charachon[56] report the case of a man, age 47 years, who was admitted to the hospital with suppuration of the left ear; there was pain but no fever. Immediate operation revealed fungous abscess of the antral zone, with spontaneous operation, and sequestration of the outer wall of the aditus. Inoculation of the fungous material produced tuberculosis in guinea pigs. Histological examination of the fungosities demonstrated giant cells, but no Koch's bacilli were found in the pus. The Bordet-Wassermann and Kahn tests, as well as the Meinicke reaction, were negative. In the third month after admission the vision became impaired, the patient was somnolent, had severe pains in the head and did not leave his bed.

Stiffness of the neck associated with these symptoms suggested meningitis. Death occurred in the fifth month.

The autopsy findings did not confirm the assumption of meningitis. The cerebrum contained a hard fungus, grayish in color, the size of a large walnut, that arose from the petrous bone; a similar smaller growth was found in the cerebellum, where it arose from the petrous pyramid. There was some infiltration of lymphocytes, but giant cells were not present. There were tubercles in the apex of the left lung; the other visceral findings were unimportant. The author points out the unusual character of this case, in that tuberculosis of the ear did not lead to meningitis.

Berblinger[57] holds tuberculosis of the tonsils is far from being the rare disease it is believed to be. In Zink's tabulation in 1930 there appear only 11 authenticated cases for the preceding 40 years. This is due to the fact that investigators usually accepted only those cases that were supported by autopsy findings. Some investigators now accept the clinical proof of primary infection if no new foci develop within several years after tonsillectomy. The clinical diagnosis of primary infection is extremely difficult. Anatomically, there is no difference between a primary resorptive and a secondary resorptive process, at least not after the beginning stage. A very detailed history of a personal case of the author's is presented with the paper.

Cuvier, Duchemin, Ringenbach and Bentéjac[58] review the literature which ascribes to the tonsils an important role in the defense of the organism. The authors investigated the question whether diseased tonsils or tonsillectomy are related to tuberculous infection. Exact conclusions cannot be drawn from the 474 cases examined, but the high percentage of cases of diseased tonsils and of previous tonsillectomies seemed to indicate the condition of these organs should never be neglected in tuberculous patients, who should be treated with particular regard to tonsillary manifestations, and that tonsillectomy should be performed in pretuberculous patients only in case of absolute necessity.

Leroux and Loiseau[59] believe primary tuberculosis of the tonsils exists, but definite proof of its primary nature will not be available until its exact frequency can be defined. This dis-

ease is latent in most instances, and of difficult diagnosis. Its presence must be taken into consideration in every case of cervical adenopathy, subacute or chronic. Histologic examination is the only method of proving or disproving its tuberculous nature. Total tonsillectomy is indicated and well tolerated, but it must be preceded by a thorough examination of the patient. Whenever the tuberculous infection centers in the tonsils, apparently without advancing beyond the first cervicoganglial stage, it constitutes a strictly localized tuberculous focus; the prognosis is on the whole good. A personal case is presented. There is no bibliography.

Roch[60] presents a detailed history of a woman patient suffering from a disease with the clinical symptoms of articular rheumatism. A most thorough examination revealed infection of the tonsils as the only focus. A bilateral tonsillectomy was performed and the patient recovered. One tonsil was filled with tubercles. The author suggests the rheumatic pains were caused by tuberculous toxins.

Barmwater[61] holds that in almost 100 per cent of all cases of sarcoid of Boeck, tuberculoid structure was found in the tonsils. In a series of 10 patients with definitely diagnosed pulmonary tuberculosis, no signs of sarcoid of Boeck were found on the skin and mucosa. The tonsils of six of the patients presented tuberculoid formations, of which five were unilateral and one bilateral. The condition of the tonsils cannot, therefore, be used for differential diagnosis between pulmonary tuberculosis and sarcoid of Boeck. Various statistics report tuberculosis of the tonsils in from 5 to 100 per cent of tuberculous patients, and 0.5 to 4 per cent in nontuberculous patients. Aage Plum, of the Finsen Institute, found tuberculous tonsils in 21.3 per cent of his patients with inflammation of the cervical glands.

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Physicians Building.

SYMPOSIUM ON THE OTOLOGICAL COMPLICATIONS OF THE ACUTE INFECTIOUS (CONTAGIOUS) DISEASES.

(a)—OTOLOGICAL CONSIDERATIONS – LANTERN SLIDES.*

DR. ALFRED SCHATTNER, New York.

In order to interpret the clinical manifestations and foresee the probable course of otitis in infancy and early childhood, the anatomy of the ear at this period, which is very different from that of the adult, must be thoroughly understood.

1. The temporal bone of the newborn is composed of three parts: The squamous, the petrous and the tympanic, which fuse together in the further development.

Petrous Bone: The cerebral surface of the petrous terminates laterally in the tegmen tympani and antri, which are in apposition to a similar plate of bone projecting from the squama, the petrosquamous suture intervening. Posterior to the middle of the cerebral surface, the eminentia arcuata projects upwards, containing the superior semicircular canal and forming the roof of a small cavity, fossa subarcuata, containing a dissepiment of dura, which later obliterates wholely or in part. This process begins at the edges of the cavity with the formation of bony ridges, the membranous contents persisting for some time, traces occasionally remaining in the adult.

At the lateral margin of the superior petrosal surface, the hiatus spurious canalis facialis is found forming a lateral opening of the Fallopian canal where the latter makes its final bend downwards and posteriorly. Later, a bony plate pushes forward over this area, forming the semicanalis nervi Vidiani for the great superficial petrosal.

At the apex, the impressio nervi trigemini presents itself. The cerebellar surface of the petrous bone, bounded laterally

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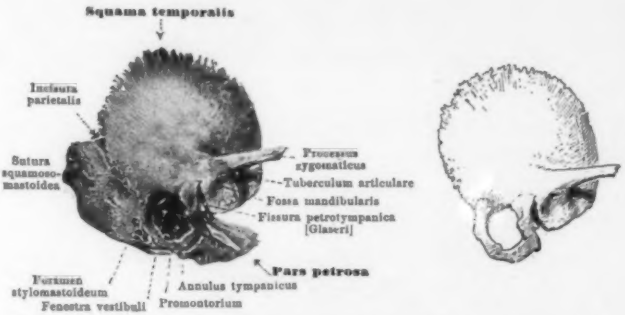
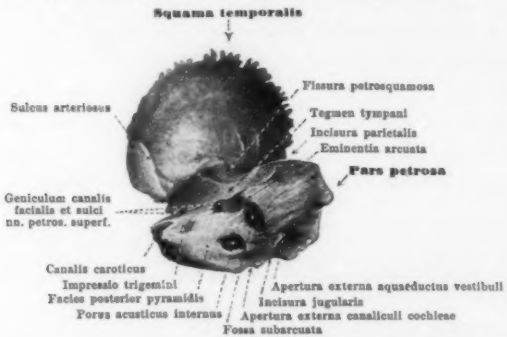
by the groove for the sigmoid sinus, presents the internal auditory meatus at its centre with its axis in the frontal plane. The internal auditory meatus is short (4 to 6 mm.), the fundus revealing the horizontal bony ridge separating upper and lower compartments. Its development is slow, reaching adult dimensions at puberty.



15 and 16. Right temporal bone, *os temporale,* from the newborn child, seen from without.

(15: complete, 16: squama temporalis and annulus tympanicus alone.)



(Seen from within.)

Fig. 1. Os temporale, newborn. (From "Hand Atlas of Human Anatomy," Vol. I, by W. Spalteholz.)

The lateral surface of the petrous is visible up to the end of the first year, the tympanic bone hiding it from then on. The fenestra vestibuli occupies its centre, its posterior and superior margin being adjacent to the Fallopian canal. Here a dehiscence is frequently patent up to the fourth year,

explaining why facial paralysis in acute otitis is found more often in infants.

2. Changes of the endocranial surfaces due to growth:

a. Closure and obliteration of the fossa subarcuata.

b. Covering of the superficially placed superior semicircular, commissure and upper half of the posterior semicircular with a 2 to 5 mm. thick layer of compacta.

c. Apposition of 3 to 7 mm. of dense bone to the upper pole of the cochlea.

d. Development of a bony plate over the hiatus spurious canalis facialis.

e. Development of a bony plate over the external opening of the vestibular aqueduct.

f. Lengthening and narrowing of the cochlear aqueduct, frequently with later fibrous stricture.

g. Marked lengthening of the internal auditory meatus with moderate constriction of its external opening.

h. Hollowing out of the sigmoid sulcus, appearance of superior and inferior petrosal sulcus and the petrosquamous sulcus, if a petrosquamous sinus persists.

3. External Ear: The external auditory meatus is rarely patent before birth, but even after exfoliation of the embryonal epidermis and detritus, the meatus is not always aerated. The softness of the cartilage and the lack of bony support cause the superior wall to be in apposition with the inferior, thereby presenting the external auditory meatus as a horizontal fissure leading from above anteriorly to below inferiorly. The bony meatus is absent, since bone has not yet developed beyond the tympanic ring.

The newborn does not present a mastoid process and the tympanic bone is present only as a grooved ring, forming three-quarters of a circle, in which the membrana tympani is inserted and to which the membranocartilaginous meatus is attached. The membranous meatus is united by thick fibrous bands to the almost horizontally placed external wall of the attic.

With the hardening and increased elasticity of the cartilage, the rudimentary lumen is further enlarged by ossification of

the membranous part of the external canal, extending laterally from the tympanic ring and taking part in the formation of the glenoid fossa. Defects in this ossification persist for varying intervals; some may remain permanently. The development of the posterior bony meatal wall is dependent upon the development of the mastoid process, which only becomes marked when the child begins to balance its head freely through the aid of the neck and head muscles, at the end of the first year of life. Rickets and retarded body development

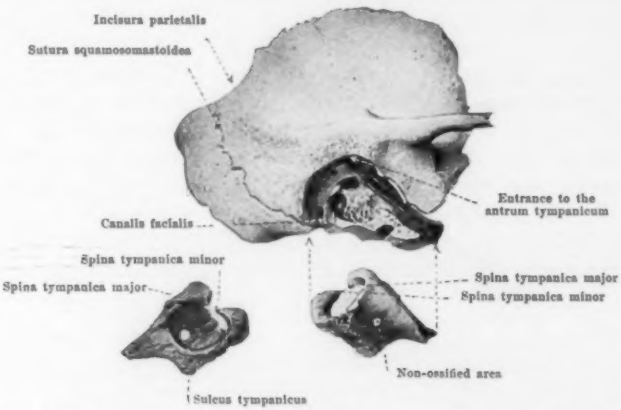


Fig. 2. Os temporale at seventh or eighth year. (From "Hand Atlas of Human Anatomy," Vol. I. by W. Spalteholz.)

may keep the mastoid process rudimentary for two or three years.

Cerumen is secreted at the end of the first week, these glands then becoming very active for several months, when they again subside to normal limits.

The membrana tympani is a direct extension of the superior meatal wall, acquiring its adult declination and inclination with the development of the bony meatus.

4. Middle Ear: The marked inclination of the membrana tympani in the newborn and during the first year of life is of pathological significance. In the adult, the posterosuperior quadrant approaches the horizontal plane more closely than the other three quadrants. This quadrant, therefore, is the recipient of the direct pressure of exudate, whereas the other

quadrants are only exposed to lateral pressure, causing the teatlike projections present in this area. In the infant, direct pressure is exerted on all four quadrants, so that these nipple-like bulges may also be found in one of the other three quadrants. A light reflex is absent on the tympanic membrane of the newborn.

The middle ear of the newborn consists of tympanum, cartilaginous tube and antrum; osseous tube and mastoid are absent. The tympanum, therefore, communicates more directly with the nasopharynx; the lumen of the tube is relatively wide and about 17 mm. in length. The pharyngeal ostium is situated at the lower margin of the choana. The antrum is adjacent to the outer cortex, often containing cartilaginous areas and, therefore, easily perforated in suppuration, leading to subperiosteal abscess. A fistula through the posterior meatal wall or perforation through the postero-superior quadrant may also be the pathway for an abscess of the antrum to evacuate itself. The stylomastoid foramen and facial are superficially placed and on the lateral surface of the skull. The squama is thin, and the temporal lobe near the bony surface.

The mucous membrane of the nasopharynx of the infant contains numerous mucous glands, lymphatic nodules and adenoid tissue.

The axis of the tympanum in the newborn is directed forwards and inwards, so that the posterior part of the tympanic cavity is much closer to the examining eye than its anterior portion.

Due to the gelatinous embryonal padding of the mucous membrane, the tympanic cavity is markedly reduced and is only enlarged with the progress of liquefaction of this tissue and pneumatization. At the end of the first year, the attic is not yet pneumatized, although meso- and hypotympanum are aerated.

The floor of the tympanum is formed by a plate of petrous bone reaching to the annulus tympanicus, fusing with the latter at a later date.

The lateral wall of the attic is a continuation of the thin plate of the bone forming the superior meatal wall and is genetically a part of the squama.

The ossicles increase 10 per cent in size from birth to adult life. The pars tensa of the tympanic membrane at birth measures 7.5 x 11 mm. In the adult, it measures 9 x 11 mm.

The circular fibres enveloping the manubrium mallei are very dense and appear as bone on examination, causing the manubrium to look thicker than it actually is. Only when marked atrophy or serous infiltration takes place, these fibres become transparent, revealing the manubrium in its actual state as a thin line.

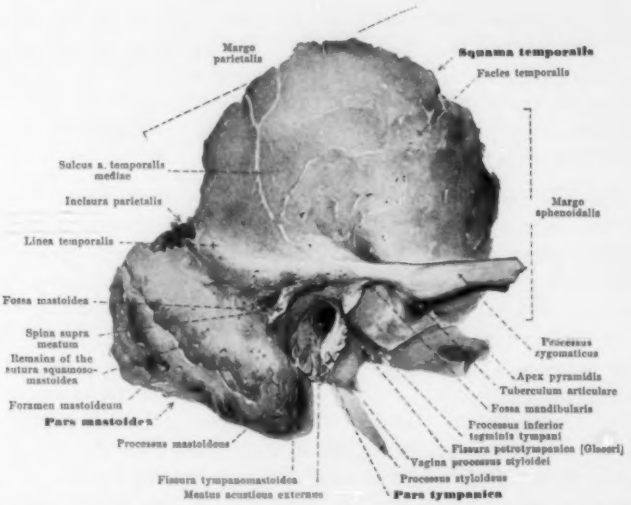


Fig. 3. Os temporale, adult. (From "Hand Atlas of Human Anatomy," Vol. I, by W. Spalteholz.)

The epithelial covering of the external surface of the drum membrane is markedly thickened, and sometimes in a state of excessive desquamation, forming nodules containing cholesterin crystals and masquerading as a cholesteatoma.

Wittmaack states that normally pneumatization is complete at the end of the fourth year; when delayed, at the end of the sixth year. Impaired ventilation of the middle ear impedes pneumatization temporarily or permanently. Albrecht points out that well developed children pneumatize more easily.

Dehiscences of the facial canal are found in the majority of infants.

The distance between the tympanic membrane and the labyrinthine wall varies. Alexander states that shallow tympanic cavities are less resistant to disease and predispose to catarrhal and inflammatory affections.

The petrosal apex is rich in bone marrow and lacks resistance to infection, predisposing it to cavernous sinus phlebitis. The defects of the endocranial cortex account for the rapid development of meningitis in early infancy.

The mucous membrane folds are the remains of the extensive mesodermal tissue cushions which fill the tympanic cavity in the fetus and newborn. With the entrance of air through the tube, resorption begins. Infection of the tympanum in the first weeks of life causes rapid purulent disintegration of this loose mucoid tissue.

The absorption of the gelatinous tissue is most rapid in the hypotympanum, but the mucous membrane remains thicker here than in the remaining tympanum, forming numerous small folds containing lymphatic infiltrations. At the end of the eighth week of life, the mesotympanum is free from gelatinous tissue. The mucous membrane is thin, the folds remain as permanent structures. The attic retains its embryonic character for some time, being traversed by a network of gelatinous tissue, which may persist to the end of the second year in cases of nutritional disturbances. Aschoff claims that retrogression of this tissue is initiated without the influence of aeration and respiration, in the fetus, during intrauterine life. Preysing claims active invasion by cellular tubes containing air. Rickets impedes these processes.

It is conceded that the anatomical stigmata characterizing the nonresistant tympanic cavity are hereditary. This will explain the hereditary nature of many acquired middle ear affections.

The infantile middle ear cavity is characterized by:

a. Short and spacious tube.

b. Markedly constricted middle ear spaces.

c. Rudimentary mastoid process.

These characteristics stamp the infantile ear as of inferior quality, a presumption of importance in the rapid and frequent occurrence of catarrhal and infectious processes in the infantile tympanic cavity, as well as the relationship of these processes to gastrointestinal affections. Otitis media and dyspepsia are closely associated in early infancy. According to Alexander, 50 per cent of all infants who died in the first

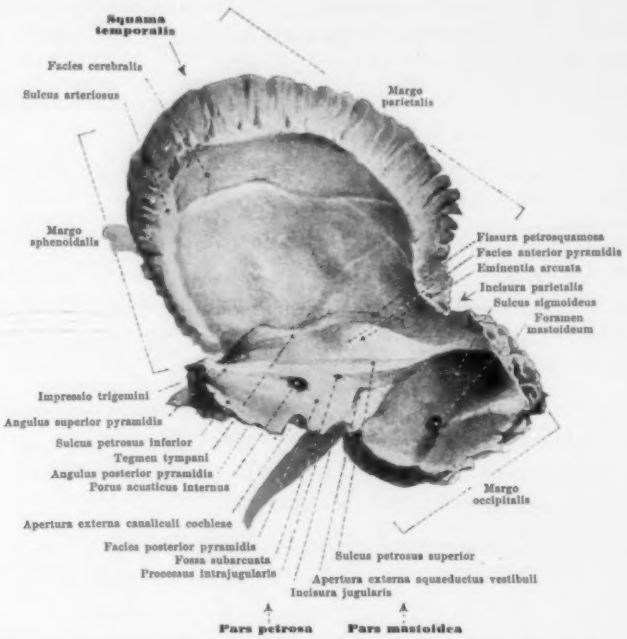


Fig. 4. Os temporale, adult. (From "Hand Atlas of Human Anatomy," Vol. I, by W. Spalteholz.)

year of life revealed this syndrome. Otitis latens with drainage through the wide open tube, and nonperforation of the drum membrane with late otoscopic evidence may be the cause of serious diagnostic error.

The mastoid process is primarily a muscular process for the insertion of the sternocleidomastoid, and its enlargement is coincident with the rapid increase in thickness of the sternocleidomastoid at a time when the infant begins to carry and

balance its head freely in space, the greatest development taking place at the end of the first year, when the child learns to walk. Diseases which delay walking also impede the development of the mastoid process. Pneumatization is rapid in the second year of life.

5. The Internal Ear: The labyrinthian capsule is an independent structure in the first weeks of life, being separated from the surrounding bone by medullary spaces. In the presence of rickets and tuberculosis, this condition may persist, favoring sequestration of the internal ear in the advent of suppuration.

From birth to the end of the second year, the following topographical conditions are present:

a. The external limb of the lateral semicircular projects from the mesial antral wall.

b. The vestibular portion of the cochlear canal is situated beneath the promontory.

c. The eminentia arcuata corresponds to the summit of the superior semicircular, the fossa subarcuata being encompassed by it.

d. The commissure of the semicirculars is plainly visible on the posterior surface of the petrous bone.

e. The upper half of the posterior semicircular forms a ridge on the posterior petrous surface.

f. The superior portion of the basal coil of the cochlea presents a convex elevation on the cerebral surface of the petrous bone.

g. The external orifices of the two aqueducts and the hiatus spurious of the facial canal present freely on the superior and posterior surfaces of the petrous bone.

In the further development of the petrous, the bony capsule of the internal ear is covered by an ever increasing layer of bone. Of topographical importance, only the prominence of the horizontal semicircular and the promontory remain unchanged. The internal ear increases 18 per cent in its postembryonal growth.

The internal ear of the infant is more directly connected with the cranial cavity than is that of the adult. The cochlear

aqueduct is short and of large calibre, as is the internal auditory meatus, whose fundus may possess a greater diameter than that of the adult. Endocranial pressure changes are directly communicated to the membranous labyrinth. The dura is more closely attached to the bone. These circumstances bring about the fact that of all the endocranial complications, meningitis develops most rapidly and is the most frequent, brain abscess and sinus thrombosis not having the necessary time to develop. The intimate connections between the inner ear and the endocranium are the cause why the internal ear is so frequently destroyed in endocranial infections of infancy. Meningism is explained on the same basis.

II.— Causes of Otitis in the Acute Infectious Diseases.

Edema of the mucous membrane in the nasopharynx, congestion due to passive dorsal posture, debility, impaired nasal respiration, and respiratory disturbances due to the accumulation of secretions in the nasopharynx, and impaired expectoration favor otitic involvement. The function of the auditory tube and ventilation of the tympanum are quickly impaired. In older children, the usual signs of tubal closure, such as fullness, feeling of weight in the head and moderate impairment of hearing, manifest themselves. With the advent of micro-organisms into the tympanic cavity per tubam, as well as through lymph and blood channels, inflammatory processes are set up directly or on the basis of catarrhal changes, leading to otitis and suppuration. The micro-organisms are sometimes identical with those of the primary infection. This is usually the case in diphtheria and influenza, almost universally in meningitis.

Wittmaack claims as a predisposing factor developmental disturbances of the middle ear, and bases the acute inflammatory hyperplasia of the mucous membrane on the persistence of the gelatinous embryonal padding.

The internal ear and auditory nerve are exposed to many diseases in the acute infections, such as purulent internal otitis, panotitis and neurotitis interna.

Otological examination is indicated in all children with an infectious disease, even though no symptoms referable to the ear may be present. This examination is especially urgent

where auditory signs are present, such as impaired hearing, headache, vertigo, disturbance of equilibrium, vomiting and so forth.

III.— Prophylaxis.

Special attention must be paid to the nasopharynx and throat. The congestion of the mucous membranes must be reduced and the respiratory pathways made patent. Mild antiseptics and aids to the removal of secretions may be indicated. Change of posture helps reduce the collections of secretions in the nasophahrynx.

IV.—Clinical and Pathological Manifestations in the Various Acute Infectious Diseases.

1. Scarlet Fever: The incidence of otitis varies with the epidemic. There is no uniformity of consideration in the statistical studies, some authors including any change in the middle ear, while others only enumerate purulent otitis. Otitis preceding the infectious disease was rarely differentiated. The reports vary from 5 to 50 per cent of involvement of the middle ear in scarlatina. We may say that purulent otological complications are very frequent in scarlet fever, but not as frequent as sometimes assumed.

The old classification of early and late forms, exanthematic and postexanthematic onset, is outdated. No definite line of demarcation has ever been agreed upon nor are the two forms clinical entities.

Clinically and pathologically, however, we may distinguish two forms:

1. Ordinary or genuine purulent otitis media, which does not differ from that found in the absence of infectious diseases.

2. Necrosing purulent otitis media scarlatinosa, specific for scarlet fever and characterized by its destructive tendencies. This type is, however, rare, comprising only 0.75 per cent of purulent otitides in scarlet. It is characterized by necrotic processes involving the deeper layers of the mucous membrane and even the bone crumbling before its onslaught. Multiple thrombi, extreme congestion and lack of round-cell infiltration are the histological picture. The membrana tympani is rapidly destroyed, often resulting in a total defect of the membrane. The ossicles are necrotic, partly destroyed,

often lying freely as sequestra in the tympanic cavity. Even the osseous walls of the tympanic cavity, including the labyrinthine capsule, are not spared. A purulent labyrinthitis is often localized, with serous labyrinthitis involving the remaining structure, but may be generalized with sequestration of the entire labyrinth.

Similar processes take place in the mastoid, almost universally causing sequestration of greater or smaller areas.

The etiology of this necrotic type is not clear and usually presents itself in severe cases of scarlet. The pathway of infection is probably hematogenous, as indicated by the simultaneous involvement of some of the nasal sinuses and the multiple thrombosis. Hemolytic streptococci are invariably found, which are also the causative factor in the nonspecific purulent middle ear infections in scarlet fever.

Clinically, the discharge is hemorrhagic in the early stages, quickly becoming fetid, and containing necrotic particles and sequestra. The extension of the process to the facial and labyrinth is by no means rare.

The otological symptoms are often overshadowed by the serious general condition, the marked deafness and labyrinthine disturbances being revealed upon the recession of the general infection.

3. There is a third type, which is less virulent and may be considered a mixed type without sharp demarcation from either of the above types. The main characteristic is the large perforation and continued discharge, whereas in the genuine type the perforation rapidly closes with the cessation of discharge. Extended necroses are absent in contradistinction to the necrosing type.

Strange as it may seem, the mortality, even of the necrosing type, is low, and the labyrinthine complications are typical in their benign character. Endocranial complications are extremely rare.

The prognosis, as far as function is concerned, is dependent upon the extent of the destructive processes. The process may heal with epidermization and adhesions, or chronic discharge with marginal perforation. About one-third of the cholesteatomata have been traced to a previous otitis in scarlet, and 12 to 25 per cent of chronic discharging ears are

caused by scarlatina. A large proportion of acquired deafmutism is due to scarlet fever.

2. Measles: Otitic involvement in measles is almost universal and is an integral part of the general infection, just as the conjunctivitis, rhinitis and bronchitis, and is of hematogenous origin, due to the unknown virus of measles. Secondary infection takes place by way of the Eustachian tube. This exanthematic otitis is often overlooked, explaining the statistical discrepancies which vary between 10 and 50 per cent. Nadoleczny, in systematic examinations, found marked middle ear involvement in 59 per cent of the cases, and purulent otitis in 13 per cent.

Purulent otitis has been observed during the prodromal, eruptive and desquamative stages, usually reaching its clinical importance due to perforation and discharge during desquamation.

The clinical picture is usually that of the ordinary O.M.P.A.; however, there are cases resembling the third or mixed type encountered in scarlet, with large perforations and tendency to chronicity, accounting for 7 per cent of chronic ears. Extensive necrosis is extremely rare. A toxic neuritis with normal membrana tympani accounts for 4 per cent of the cases of acquired deafmutism.

In view of the prevalence of measles and the few reports of permanent injury to hearing or complications, the prognosis must be considered as very good.

3. Diphtheria: Purulent affections of the middle ear are relatively rare.

The distinction between secondary otitis and affections due to diphtheria bacilli is very difficult, due to the uncertainty of identifying and excluding pseudodiphtheria bacilli and the frequent presence of mixed infections. Only in rare cases with pseudo-membrane formation and destructive processes is the clinical picture clear.

Primary involvement of the ear reveals the absence of a clinical lesion in any other portion of the upper respiratory tract even though bacilli may be found.

Secondary involvement presupposes a primary clinical lesion, usually in the throat.

The two clinical entities are:

1. The rare typical diphtheritic lesions with the formation of pseudomembranes. The membrana tympani is discolored, yellowish-gray and covered with membranous exudate, sometimes extending into the external auditory meatus. The discharge is profuse, serous, and destruction of the tympanic membrane and middle ear may develop rapidly, the same processes taking place in the mastoid bone. Involvement of the labyrinth, however, is very rare.

2. Otitis of the genuine type is the usual picture with diphtheria bacilli being present. The course is subacute, marked pain and more frequent involvement of the mastoid process are characteristic.

The prognosis of the membranous type is grave, being a part of a severe toxic-diphtheritic process open to secondary infection with streptococci. Restitutio ad integrum is impossible, and a chronic discharging ear with the formation of a cholesteatoma at a later date is frequent. Early administration of serum is essential even before the diagnosis has been substantiated.

Toxic involvement of the auditory nerve is rare.

4. Influenza: The otitis is here secondary to the general infection, which lowers the tissue resistance to the bacterial onslaught.

The incidence of otitis increases sharply with the prevalence of the grippe, but varies greatly in different epidemics and bears no definite relationship to the severity of the general infection.

Pfeifer's bacilli are rarely found, the usual micro-organisms predominating.

The typical form is the hemorrhagic type, which often accounts for over 50 per cent of the cases, and would probably be diagnosed more often if early examination were made before perforation and discharge have taken place.

Pathological Anatomy: Marked hyperemia with extravasation of blood in the tympanic cavity, submucosa and subepithelial spaces, causing blebs on the drum membrane and external meatus.

Clinical Course: Only the early stages are typical, since we are considering different infections following one general

disease. The pain is characterized by its severity due to irritation of the nerve fibrils ending freely in the intraepithelial layers. The discharge is hemorrhagic, gradually becoming purulent, at which stage the picture becomes that of the ordinary O.M.P.A. Severe cases resemble the necrotic type of scarlet fever. In rare instances fatal endocranial complications develop with great rapidity, labyrinthitis and meningitis being found at autopsy without destruction of bone in the tympanum or mastoid. The prognosis quoad vitam is graver than in the ordinary otitis, measles or scarlet, but better as regards function and repair of the auditory organ. Persisting discharge and perforation are extremely rare, and, even more so, involvement of the cochlear nerve.

5. *Epidemic Cerebrospinal Meningitis:* One-third of all the cases of acquired deafmutism is due to meningitis. Hematogenous propagation is essential for understanding the auditory involvement. The infection in the arachnoid spreads along the nerve sheaths, destroying the neuroepithelium very quickly. Almost without exception, the infection of the internal ear is diffuse and bilateral, the structures being replaced by granulation tissue, which later becomes fibrous and may ossify. The middle ear may be infected secondarily per tubam, metastatically by way of the blood stream or by continuity from the labyrinth. Due to the general condition, the injury to the ears may not become manifest until the meningitis clears up; in other cases, the attack upon the labyrinth is revealed in the development of nystagmus, vertigo and vomiting. Early administration of polyvalent sera and chemotherapy are indicated to forestall auditory involvement.

6. *Parotitis; Mumps:* The affection of the internal ear is due to infectious-toxic action of the mumps virus by way of the blood or lymph channels. It may attack the end-organ or the nerve, sometimes including the facial. The disease is usually unilateral and of sudden onset, with labyrinthine symptoms. Recovery, due to the serous nature of the lesion, is the rule; but impaired hearing, even complete loss of hearing, may be a sequel.

7. *Other infectious diseases* may involve the organ of hearing, but are of less practical significance. Among the more important may be named: Typhoid fever, pertussis, erysipelas and polyarthritis.

V.—Treatment.

Drainage, with an extensive myringotomy as early as possible, is the cardinal principle. In the destructive types of infection, early surgical interference is indicated to limit the spread of the disease. Otherwise, we shall be guided by the surgical principles as applied to ordinary otitic infections.

VI.— Conclusions.

The clinical picture of otitis in the more important infectious diseases has been described with a view to recognizing certain types characteristic of the concomitant infectious disease, on which basis therapeutic principles are applied, dealing with the pathology present.

115 East 61st Street.

AMERICAN OTOLOGICAL SOCIETY, INC.

The Seventy-third Annual Meeting of the American Otological Society will take place on May 30 and 31, 1940, at the Westchester Country Club, Rye, N. Y., under the presidency of Dr. Horace Newhart.

Attention is directed to the fact that May 30 is Decoration Day, and the Club will provide many forms of entertainment. In addition, there will be a golf tournament between the members of the Laryngological Association and the Otological Society on May 29. Players will be divided into Classes A, B and C, according to their handicaps. It would be wise, therefore, for players to be supplied with certified handicaps from the club of which they are members.

The guests of honor of the Society will be Dr. Thomas J. Harris and Dr. Frederick L. Jack. After more than 20 years of service, Dr. Harris has determined to relinquish the position of Secretary-Treasurer.

SYMPOSIUM ON THE OTOLOGICAL COMPLICATIONS OF THE ACUTE INFECTIOUS (CONTAGIOUS) DISEASES.

(b) — ACUTE CONTAGIOUS DISEASES – UPPER RESPIRATORY COMPLICATIONS.*

DR. ARTHUR S. WILSON, New York.

We consider here the rhinologic and laryngologic complications of contagious disease as seen at the Willard Parker Hospital during the past 12 years. Most of them will not be new to this audience, but some, I hope, will be interesting.

These complications are seen chiefly in scarlet fever, diphtheria and measles, and scarlet fever is responsible for most of them. These complications consist of pharyngitis, tonsillitis, peritonsillar abscess, cervical adenitis, retropharyngeal abscess, laryngeal abscess and sinusitis.

As for the incidence of these complications as affected by sulfanilamide, my impression is that they are less, but this year the streptococcus infection is mild, and statistics as yet are not of much use, because there has not been enough time or enough cases to evaluate this drug properly. The men in charge of contagious diseases in general have not made up their minds as to the effectiveness of sulfanilamide.

We see abscess of the larynx and papilloma of the larynx because they are mistaken for diphtheritic croup in the absence of a direct examination of the larynx.

The sinusitis in scarlet fever is acute and severe, and the symptoms are well known and do not require mention. The treatment consists of ephedrine preparations introduced into the nose, and then catheter suction, particularly in infants and young children. Orbital abscess from ethmoid or frontal sinus involvement is not uncommon, and operation — either external or internal — is indicated. Several cases have recov-

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ered well after the internal operation, but I personally prefer the external route because it is adapted to all cases. It is an open operation, and all necrotic bone can be observed and removed; the ethmoids, sphenoids and antra are always opened at the same time. Operation should be performed early. The temperature or the contagious disease is no bar to operation. These cases should be treated carefully after they recover from the acute condition. Consider a chronic running nose as bad as a chronic running ear. I believe that contagious disease with sinusitis in childhood is the origin of many cases of chronic sinusitis. The usual care must be taken that infection is localized before operation, and I believe that the chief value of sulfanilamide is that it hastens the process of localization.

In this audience, it seems unnecessary to do more than state that tonsillitis, pharyngitis, adenitis and retropharyngeal abscess exist and are treated as usual. Retropharyngeal abscess is often missed and tracheotomies are done unnecessarily because there is respiratory obstruction and the patient's larynx is not carefully examined with a laryngoscope.

In measles, sometimes an eruption is present in the larynx. In diphtheria, we have a swelling in the larynx with the presence of a membrane. Dr. Lynch, many years ago, did a lot of bronchoscopic work and used to remove this membrane from the trachea and bronchi. We very rarely use a bronchoscope. I think we see many more of those cases because cases of laryngeal diphtheria are treated earlier than they used to be.

Our worst cases, and the most difficult to treat, are, strictly speaking, not complications of contagious disease. I refer to laryngotracheobronchitis. These cases we see at Willard Parker because of one symptom — respiratory obstruction. Inasmuch as these cases are not usually laryngoscoped, cases with respiratory obstruction are referred to Willard Parker as "croup," with the possibility that they are laryngeal diphtheria. These cases as we see them are always severe and dangerous, because of respiratory obstruction, and because only the obstruction cases are sent to us. In other words, we do not see the many mild cases of laryngotracheobronchitis as seen by the general practitioner and pediatrician.

These patients are usually young children and infants, and the younger the child the more severe the symptoms and the greater the mortality. The streptococcus is the most frequent bacteria found, but the pneumococcus and staphylococcus are frequently present.

The symptoms consist of temperature, malaise, cough and laryngeal obstruction. The pharynx is red and congested, the larynx red, swollen and congested, and the tracheal mucous membrane is swollen and red, with mucopurulent exudate, but there is no membrane — and by a membrane, I mean a distinct conglomeration of epithelium and mucopus which adheres to the bronchus.

We describe the laryngeal obstruction as superglottic and subglottic, by which we mean that the obstruction is above or below the cords. Both may be present, and usually are. We usually intubate these cases immediately. The opening of the tube is frequently blocked by the swollen false cords and arytenoids or by exudate. If the mucous membrane below the lower end of the tube is much swollen, intubation gives little or no relief, and tracheotomy gives only a partial relief, because the entire tracheobronchial mucous membrane is swollen and masses of thick secretions partially or completely block the small airways of the children's tracheae and bronchi. At present, we are using the direct method of intubating and extubating.

We consider as indicated for tracheotomy those cases in whom obstruction is not relieved by intubation, and cases whom we cannot extubate after one week. This statement needs some explanation. In diphtheritic croup, the patients recover or die within a few days, and an intubation tube is inserted for only a few days. In laryngotracheobronchitis, the patients do not recover so rapidly, although they frequently die rapidly, and, therefore, they require help in breathing for about one or two weeks or more. The pressure of an intubation tube upon a succulent, tender, inflamed mucous membrane produces ulceration, and when these are deep the resulting scar formation causes chronic laryngeal obstruction.

Some years ago we adopted a plan of tracheotomizing these tube cases, after an arbitrary period of two weeks' intubation.

We have now reduced that time to one week. This is controlled by daily laryngoscopic examinations, and if ulcerations develop we perform tracheotomy immediately.

Some of these patients repeatedly cough up their tubes between the fourth and seventh day, and these cases are also immediately tracheotomized, because the trauma produced by frequent intubation tends to produce ulceration, and the patients become exhausted. Intubation is suitable for short cases but not for prolonged cases, and, of course, presupposes a resident staff trained in intubation. This plan has abolished chronic intubation cases at the Willard Parker Hospital.

I read a paper some years ago on the subject of "Chronic Laryngeal Obstruction" and the prevention of chronic intubation cases, and I believe that the careful laryngoscopic examination of the larynx and tracheotomy at the slightest sign of trauma or ulceration of the larynx is the key to success in the prevention of chronic laryngeal obstruction in these cases.

In laryngotracheobronchitis, cyanosis is not common because the obstruction does not occur suddenly or completely, but is slow in development and incomplete. In these cases of slowly progressive obstruction of the larynx there is a high pitched inspiratory rasp, accompanied by retraction of the supraclavicular region, the spaces between the ribs and the substernal region. The face is pale and not cyanosed, the lips are pale, and as the dyspnea becomes more severe the pallor of the face increases and a white line appears on the upper and lower lips, completely surrounding the mouth. This is a terminal sign indicating impending death through exhaustion and lack of oxygen, and is an imperative indication for immediate tracheotomy.

Sudden death occurs following tracheotomy, both in adults and in children, by the sudden dilution of the residual carbon dioxide in the lung by the inrush of air. The remedy is the administration of carbon dioxide and oxygen, and this should be on hand and ready for use alongside of the operating table. It is best to administer carbon dioxide and oxygen to the patient as a routine immediately following a tracheotomy, because if one waits for symptoms, the patient has already ceased breathing from lack of carbon dioxide, and it is then

difficult, if not impossible, to get sufficient carbon dioxide to the respiratory centre in time to revive it.

Postoperative Care: The air is moistened by a pledget of gauze over the mouth of the tube. This is kept wet with normal saline. The patient is put in the Trendelenberg position so that gravity will help drain the mucopus from the lower bronchi into the trachea, from which it can be removed with suction through the tracheotomy tube, or by a bronchoscope inserted through the tracheotomy wound.

We sometimes instill normal saline into the tracheotomy tube to dilate the exudate, and we also use a weak solution of ephedrine for the same purpose. Blood transfusions are extremely valuable, and we use small quantities repeated for three or four weeks in succession.

We have used sulfanilamide persistently and as yet are doubtful of its value. The indications of treatment are to combat the toxemia and to drain the tracheobronchial tree mechanically by suction through the tracheotomy tube, or through the bronchoscope.

Complications of Tracheotomy — Hemorrhage: I have never seen a primary hemorrhage, but I have seen one of two cases of secondary hemorrhage during the second or third week as a consequence of deep infection.

Mediastinitis is rare, and usually terminates in hemorrhage from erosion of one of the larger vessels.

Emphysema and Pneumothorax: This is common, occurring in about 75 per cent of the cases; when pneumothorax occurs on both sides, it is exceedingly dangerous. It may occur spontaneously, with or without a tracheotomy.

I had a case which I would like to mention briefly because it contains all the elements of these cases: A child, age 2 months, ill for two days, was admitted with respiratory obstruction. We did a tracheotomy after the patient's parents were convinced that it was necessary. About 3:00 o'clock one morning, the house surgeon called me and said that the child was doing pretty well, but was looking pale. We bronchoscoped the child and got out a considerable amount of muco-

pus. He called up at 1:00 o'clock the following night to say that the child was in terrible condition. He had the typical white face, though no white line. We again bronchoscoped him, after which he was very much improved, though not completely improved. At 9:00 o'clock, I was called again, and the child almost died under my hands from respiratory obstruction. We put in a bronchoscope and got nothing out; we removed the tracheotomy tube and in distraction started artificial respiration, and out came a lump of dark brown, waxy stuff, and the child completely recovered.

140 East 54th Street.

SYMPOSIUM ON THE OTOLOGICAL COMPLICATIONS OF THE ACUTE INFECTIOUS (CONTAGIOUS) DISEASES.

(c) — INTERSTITIAL EMPHYSEMA OF THE LUNG WITH PNEUMOTHORAX AND SUBCUTANEOUS EMPHYSEMA.*

DR. MORRIS STERN, New York.

A case of laryngotracheal-bronchial diphtheria was presented in which a left pneumothorax and subcutaneous emphysema developed three days after tracheotomy.

The necropsy revealed no mediastinal emphysema. The right lung was adherent throughout as a result of an old pleuritis. There was a pneumothorax on the left side. The left lung was partially adherent to the chest wall and showed a partial collapse and bronchopneumonia.

Microscopic sections from the left lung revealed air spaces in the interstitial septa.

Willard Parker Hospital.

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SYMPOSIUM ON THE OTOLOGICAL COMPLICATIONS OF THE ACUTE INFECTIOUS (CONTAGIOUS) DISEASES.

(d)—EAR, NOSE AND THROAT COMPLICATIONS—LOWER RESPIRATORY.*

DR. JESSE G. M. BULLOWA, New York.

Demonstration of charts showing the occurrence of lower respiratory infections as a complication of acute infectious diseases at Willard Parker Hospital during a five-year period from 1933-1937.

Slide 1: Pulmonary and upper respiratory infections in contagious diseases, the pulmonary infections, including tracheitis, bronchitis, lobar and bronchopneumonia; and the upper respiratory infections, including sinusitis, pharyngitis, tonsillitis and croup. Among 27,652 admissions to the contagious diseases at Willard Parker Hospital in five years, lower respiratory infections occurred most frequently in 25 per cent of the pertussis cases, 12 per cent of the obstructive laryngitis, and almost 10 per cent of the measles cases. The mortality in the pneumonias of diphtheria was highest, 44 per cent; next in poliomyelitis and in obstructive laryngitis.

Slide 2: Incidence of pneumonia in measles cases. In four measles years pneumonia occurred in measles patients, in one hospital, 8.4 per cent, and in the other, 10.6 per cent.

Slide 3: Comparison of mortality rate of pneumonia in measles cases in another hospital and in Willard Parker. The mortality rate in Hospital 1 was 14 per cent; in Hospital 2 it was 23.3 per cent. We looked for an explanation of the much lower mortality rate at Willard Parker, and possibly found it in the amount of oxygen used in the two institutions. In 1935, Hospital 1 used 518 cylinders, and Hospital 2, 504. During the first six months of 1936, Hospital 1 used 400 cylinders,

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and Hospital 2, 390. In the year when there was the very marked reduction in the mortality rate at Willard Parker, there was a marked increase in the oxygen used, from 300 cylinders to 800, while in Hospital 2 only 600+ were used. It is very possible that the greater use of oxygen in these children was responsible for the lowered mortality.

Slide 4: Pneumonias in contagious diseases, Jan. 1, 1938, to July 1, 1938.

In regard to the age distribution, the incidence of pneumonia was greater in children under 2 years of age. As a matter of fact, between the ages of 2 and 6 years it was greater in Hospital 1 than Hospital 2, and then there was a marked fall in the incidence in both hospitals between the ages of 6 and 12 years, the incidence becoming insignificant over the age of 12 years. We also studied the mortality by ages, and you will notice that in Hospital 2, where there was a higher death rate under 2 years of age, there was also a higher incidence in that period, and this relationship always held, excepting during the period of greater oxygen consumption, where the mortality was less, although the incidence was greater.

We studied the incidence of lobar and bronchopneumonias in the two hospitals, and a very curious thing developed. We found the figures so entirely divergent that it isn't likely that it was a true difference. This only serves to emphasize the importance of not trying to differentiate pneumonias in children according to location. It is much more important to differentiate according to etiology.

(Demonstration of charts showing results of treatment by sulfapyradine, and a comparison of the results of treatment by sulfapyridine and serum.)

These figures are not altogether satisfactory because the cases were not alternated, and they are too few in number from which to draw conclusions.

62 West 87th Street.

SYMPOSIUM ON THE OTOLOGICAL COMPLICATIONS OF THE ACUTE INFECTIOUS (CONTAGIOUS) DISEASES.

(e)—POLIOMYELITIS.*

DR. PHILIP M. STIMSON, New York.

Dr. White has asked me to discuss in the few minutes at my disposal some of the points about poliomyelitis which otorhinopharyngotracheolaryngologists might find interesting, suggesting particularly that the items covered might include the early diagnosis and some of the treatment, and to this I think I will add certain aspects of prophylaxis.

No one can know whether or not we shall have an epidemic of poliomyelitis in New York this summer. All we can say is that when there is an epidemic it gets under way early in July, reaches its peak in mid-August, and, so far as new cases are concerned, is pretty much over before the end of September. If we were to delve into the statistics, we would find that in New York since 1930, the even summers have been very light, but the odd years have been heavy. The heaviest even summer — 1932 — had 90 cases, while the lightest odd year had some 200. In 1931, and 1935, there were bad epidemics. This is an odd year, and one might reason that an epidemic is due, but such statistics, however, mean nothing in the epidemiology of poliomyelitis.

The term "poliomyelitis" is much preferred to "infantile paralysis," because the disease no longer occurs predominantly in the infantile group, and probably only a very small proportion of those infected develop paralysis. Incidentally, there seems to be a general misconception of the meaning of "polio." I have been told a number of times that it means "soft." Actually, of course, it means "gray."

The terminology of the types of the disease has been very confusing, particularly for those cases that show meningeal

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symptoms but which do not go on and develop paralysis. They have been called preparalytic cases, meningeal cases, etc. Levinson and Harmon, of Chicago, suggested the terms "early acute poliomyelitis without paralysis," "acute poliomyelitis with slight, moderate, severe or bulbar paralysis of such and such muscles," and then when the fever is gone, a case should be called "chronic poliomyelitis with paralysis of such and such muscles." There ought to be some standard way of describing these cases so that when hospitals report results, their grouping will coincide with the corresponding grouping of others.

The disease cannot be diagnosed nor its presence suspected until some of the so-called meningeal symptoms have taken place. The incubation period is probably about seven to 10 days, during which there may be a day or two of slight fever and perhaps slight upper respiratory or digestive symptoms. These prodromal manifestations are thought to be due to the progress of the virus from the olfactory tract to the hypothalamus and thalamus, and thence to the spinal thalamic tract. When the virus progresses on to the medulla or down the cord to the meninges and the cord substance, acute symptoms of the disease develop, and when the anterior horns are sufficiently involved, paralysis appears. Finally, when the virus becomes inactive, the disease reaches the chronic stage; however, except in that very small proportion of cases in which paralysis develops, the virus will not travel all that distance to the anterior horn cells, so that many patients, while infected with the poliomyelitis virus, give no clinical evidence of the disease and cannot be diagnosed as having had it.

The only way we know this is that we find many more people whose blood will neutralize the virus than give a history of having had the disease. A number of children have had slight upper respiratory disturbances and perhaps slight digestive upsets, but no central nervous system symptoms. Yet, later it can be demonstrated that their blood is able to neutralize the poliomyelitis virus.

A true diagnosis can be made as follows: The mother reports that the child has had a fairly sudden onset of headache, moderate fever, perhaps vomiting, and pain in the neck.

The child seems pretty sick, much sicker than the degree of fever would seem to justify. He is prostrated, apprehensive and wants to be let alone. The neck and back are tender and stiff. To test that, you merely have to ask the child to sit up and touch his chin to his knee. This he cannot do. When asked to sit up in bed, the child will sit up, holding his back rigid with his arms braced behind him, quite characteristically. If you give the child a glass of water to hold, he is apt to develop quite a tremor. The reflexes are irregular, and signs of meningeal irritation, such as those of Kernig or Brudzinski, may or may not be positive. A lumbar puncture is indicated to relieve the headache, but more particularly to allow a spinal fluid examination to be made in order to confirm the diagnosis. The spinal fluid is abnormal only for about six to eight days. The fever lasts from six to 10 days, but the virus remains active over a longer period.

Poliomyelitis is probably not present if there is irrationality or coma, if there are convulsions, if there is very much fever, if there is severe pain or any swelling of the affected extremities, if there is cervical lymphadenitis or if there is prolonged illness preceding paralysis. In the presence of an epidemic, or even without it, any doctor should be on the watch for early cases.

In the prophylaxis of the disease there are about four principles or facts which have to be borne in mind. First, the virus enters via the olfactory area of the upper part of the nose, but it may enter by other routes. Certainly, in unusual circumstances it may enter through the tonsillar area before, during or after tonsillectomy. The second fact is immunity in poliomyelitis depends on the tissue resistance of the central nervous system rather than on a humoral resistance — that is, on antibodies in the tissue fluids. The third fact is that in severe epidemics, only one or two people per thousand of the population are *known* to have the disease, though probably many more have undiagnosable subclinical or abortive attacks. Fourth, there is no test for susceptibility to the disease. We have only a test for virus-neutralizing property in the patient's blood, and each test requires a monkey and is, therefore, expensive. But at that, we don't know whether the presence of neutralizing bodies constitutes immunity to the disease or not.

Methods of Prophylaxis: First, about sprays. Zinc sulphate sprays cause a loss of smell, they are very painful to administer, they may cause ulceration and necrosis of the olfactory mucous membranes and adjacent tissues, and as these preparations have been used by spraying with atomizers, they have not been successful in providing invariable protection. Failures have been reported. The mixture of aluminum sulphate and picric acid has been reported as causing sinusitis, headache, etc. Reports of its use have covered administration by atomizers only. Neither method has had the benefit of the so-called Proetz position with the head inverted.

Apropos of that position, I want to hark back to during the war, when for a time I was in the Contagious Disease Hospital at Rouen, run by the British. Charles J. Martin, the Director of Laboratories, was a very canny Australian pathologist — a delightful man. I went over to the laboratory one day and found three British soldiers lying across a laboratory table with their heads upside down, and I asked Col. Martin what he was doing. He said the three men were meningococcus carriers, and he was trying to flood their heads with a dye to see what it would do to their meningococci. Two of the three were cured with that one application. I don't know what happened to the third.

I was talking today with Dr. Josephine Neal, who has probably seen more poliomyelitis than anybody in the country, and she said she was not willing to advocate the use of sprays at this time. Dr. Sidney Kramer, of Brooklyn, when he heard about this meeting tonight, said he would like to see a large experiment done with the Armstrong preparation of aluminum sulphate and picric acid in the proper manner with the head in the inverted position, so that the nasopharynx could be flooded, and he asked me if I would ask those present what they would think about such an experiment. So much for sprays!

The second method of prophylaxis is the use of convalescent serum. In the first place, it is not practicable, because we have no test for susceptibility. We don't know to whom we have to give the serum. Secondly, we don't know how much we have to give; and thirdly, we don't know whether it is going to give a tissue immunity or humoral immunity.

The third prophylactic possibility is the postponement of tonsillectomies at the time of a poliomyelitis epidemic. It has been shown by a number of authors that a very fair number of patients whose tonsils have been removed during the time of poliomyelitis epidemics have within 10 to 14 days developed poliomyelitis, usually of the bulbar type, with a very high mortality. Sabin found that in monkeys he actually had to inject the virus into the tonsils to get that type of infection, but he and various others who have investigated this subject all recommend that in the presence of a poliomyelitis epidemic it is probably just as well not to take tonsils and adenoids out if you can avoid it.

When there is a poliomyelitis epidemic, what are we going to tell parents they can do to prevent their children from getting it? First, there is no sense running away from it, for it follows somehow or other, and the chances of their children developing the disease in a recognizable form are less than one in a thousand. It is a very bad epidemic where even one in a thousand gets the disease. Second, it is desirable to protect the integrity of the nasal mucous membrane. The mucus that coats it is a good thing to have present. Therefore — and I would like to know what you think about it — it seems desirable to keep water out of the child's nose. If he wants to go in swimming, he might wear nose clips, such as are used in sinusitis. Finally, it is well to advise that excessive physical fatigue be avoided. There have been two different articles, one of them from Germany in a large academy for boys, where they showed that a very considerable proportion of the boys got physically exhausted, and this group had a larger incidence of poliomyelitis than the other group.

When poliomyelitis does occur, what phases of the treatment particularly concern the ear, nose and throat specialist?

In the first place, as far as we know now, the most important thing in the treatment of an early case of poliomyelitis is rest — not only of the affected part, if paralysis has developed — but rest of the entire child. Some of us even think it is more important to leave the child quietly at home than to move him to a hospital. Rest is very important, and if any specialist has to examine the child, it should be done with as little disturbance as possible.

The type of poliomyelitis that particularly concerns the ear, nose and thoat specialist is one where there is pharyngeal paralysis, which is one of the manifestations of the bulbar type of the disease. We have learned by bitter experience at Willard Parker Hospital that if there is respiratory embarrassment and you put these bulbar patients in a respirator, they almost invariably die. Where there is bulbar involvement, keep them out of a respirator. Mucus is sucked down the trachea and they are asphyxiated or develop pneumonia. These patients should lie on the chest with the head to the side and the foot of the bed elevated, and fairly constant aspiration should be used.

Feeding is of considerable importance. If there is simple palatal paralysis only, these patients can usually swallow soft solids. Fluids will be regurgitated through the nose. If, when food is offered, the patients vomit, it gives evidence of involvement of the muscles of swallowing. These patients can be fed by gavage or through a Levine tube passed to the stomach. If there is choking or coughing, indicating that some of the food has gone into the larynx or trachea, nothing should be given by mouth at all. Finally, it is well to try to have the patient cough, and if there is a diminished ability to cough, you know the child is going to need aspiration, and nothing by mouth should be given. Not even gavage should be attempted, but feeding should be by hypodermoclyses, rectal drip, etc., sometimes even for two or three weeks.

There is a patient at Willard Parker at the present time who, if we didn't have the history, would seem to be a clear illustration of this type of paralysis. She has weakness of half of the face, paralysis of half of the palate, great difficulty in swallowing, a fairly complete diaphragmatic paralysis, and also some weakness of the legs. But we have a history that, six or seven weeks ago, the child had a bad sore throat, was not given antitoxin, and now she has a postdiphtheritic paralysis. To confirm the diagnosis, she also has a ciliary paralysis of the eyes, which you get in diphtheria and not in poliomyelitis. That child is on her side, is having fairly constant aspiration and is being fed through a tube.

Willard Parker Hospital.

IS THE X-RAY EXAMINATION OF THE MAXILLARY SINUS SUPERIOR TO THE SIMPLE TRANSILLUMINATION?*

DR. RICHARD WALDAPFEL, Grand Junction, Colo.

Since Heryng, just 50 years ago, performed the first transillumination of the maxillary sinus, his method belongs to the daily examination methods of the rhinologist. We use it as first orientation about the condition of the sinuses, and we are accustomed to perform it routinely as a simple and inexpensive method of diagnosis, substituting it for the more expensive X-ray. We assume that both show the same if they are positive; namely, a decrease in the air content of the maxillary sinus.

We give the preference to the X-ray because of its more exact and, in most instances, a more definite method of diagnosis. For, in the simple transillumination a difference in darkness of the maxillary sinus can be caused not only by a disease of the sinus but also by: *1.* asymmetry of the two sinuses; and *2.* varied thickness of the bone; these physiological anomalies do not play a part in the X-ray examination and do not influence the results.

That, however, even in symmetrical maxillary sinuses, and both sides normal and having equally thick bony walls, the transillumination and X-ray may furnish different findings, and that these differences of the findings may be well used for building up the diagnosis shall be shown in the following observation and discussion.

A patient, N. B., age 11 years, fell down three hours previously, face downward, on a rock and has hit the right upper jaw. She has bled a little from the right naris and has suffered some erosions on the hands and legs; she is referred by the pediatrician, Dr. R. J. Groom, with the possible diagnosis of a fracture of the upper jaw.

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*Presented before the Colorado Otolaryngological Society, Colorado Springs, May 6, 1939.

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The first examination shows the following picture (see Fig. 1): Swelling moderate of the skin without discoloration, subcutaneous edema of the right side of the face, especially noticeable below the medial angle of the eye, reaching down to the nose and above the upper lip, where a superficial erosion is to be seen. On this place the upper lip is slightly swollen on the inside also. No discoloration of bones nor crepitation to be stated. Rhinoscopically, there are some blood clots to be seen on the floor of the nose (otherwise negative). The only point which eventually would have indicated an injury of the sinus wall was a statement in the history of a

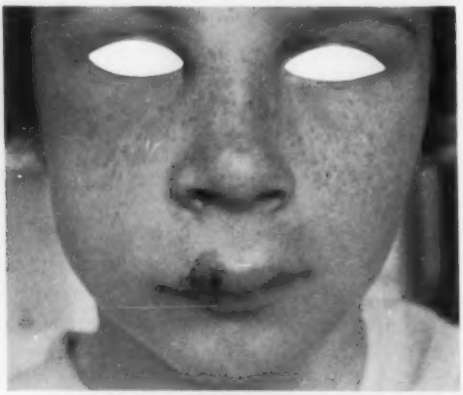

Fig. 1. Patient shortly after the trauma.

hemorrhage from the right side of the nose. In order to get information about the condition of the right maxillary sinus and its aerial content, a transillumination was done, which showed an impressive picture (see Fig. 2). The whole right maxillary sinus is unquestionably cloudy.

This finding is apt to cause an assumption of a blood filling into the right maxillary sinus and make the diagnosis of a fracture still more likely. To confirm this diagnosis, an X-ray was done. It shows (see Fig. 3) surprisingly a contrary finding. Both maxillary sinuses are symmetrical and have equally thin walls. Both maxillary sinuses show about the same air content, normal for all intents and purposes. A frac-

ture is not in evidence, and a series of X-ray conclusively proves that a fracture was not present.

A puncture of the maxillary sinus was purposely not done in order not to infect the sinus.

We are concerned here with a case of injury of the upper jaw, with suspicion of a fracture, in which the transillumination and X-ray of the maxillary sinus show quite a different picture; the transillumination showing a positive cloudiness, and the X-ray entirely negative. Asymmetry or difference in thickness of the bony walls can be ruled out as reason for the transillumination result; there remains only one explana-

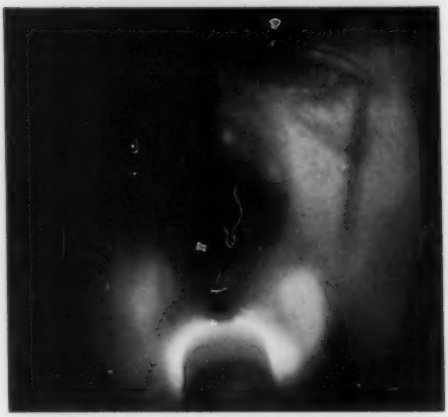

Fig. 2. Transillumination. Right side completely cloudy.

tion, that a slight swelling of the skin and subcutaneous tissue infiltration are sufficient to cause this highly positive finding (indeed, within 10 days this positive illumination disappeared according to the decrease of swelling of the skin, which within this time shows slight discoloration below the eye and above the upper lid).

The observation here discussed is interesting and allows the following statements:

1. Apart from disease of the maxillary sinus itself, darkness in transillumination can be caused not only by thickness of the bony walls and asymmetry of the maxillary sinus,

but as well and still much more by infiltration of the soft parts, even of slightest degree. In such cases the transillumination shows more than the X-ray.

2. Complete or massive cloudiness of a maxillary sinus in transillumination with negative X-ray findings is to be regarded as significant for infiltration of the soft parts, subcutaneous edema or hematoma (even if no considerable swelling or discoloration of the skin is to be seen).

Both above statements represent an addition to our usual knowledge in the diagnosis and differential diagnostic procedure in affections of the maxillary sinus.

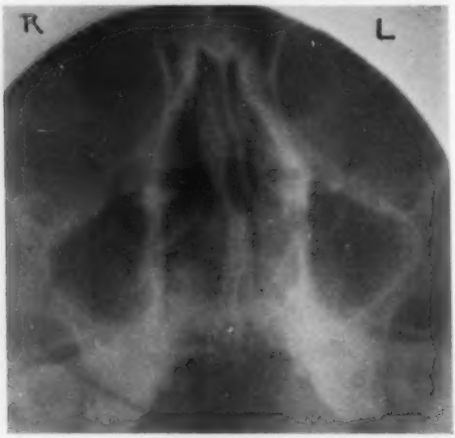

Fig. 3. X-ray of the same patient. No marked difference between right and left side.

The axiom that X-ray of the maxillary sinus is superior to the simple transillumination must be denied in certain cases, as the one described, wherein the transillumination is definitely superior.

Not transillumination *or* X-ray, but transillumination *and* X-ray have to be done in every doubtful case; they do not substitute one for the other, but complete themselves as substantiating evidence.

Both may show different details, and this difference is supporting the differential diagnosis of the diseases of the maxillary sinus.

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STUDIES OF THE VIIIth CRANIAL NERVE OF MAN.

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INTRODUCTION.

On general principles, it may be assumed that sooner or later numerical data on the number of nerve fibres in the VIIIth cranial nerve will be of some importance in both normal functional considerations and in special neuropathology. The immediate reason for undertaking an enumeration of the fibres in individuals with apparently normal hearing was the discrepancy between the number of ganglion cells in the spiral ganglion and the supposed number of fibres in the cochlear division of the so-called nervus acusticus or VIIIth cranial nerve.

Guild[1] found from 23,193 to 39,114 (average, 29,024) cells in 10 ears of young adults known by audiometer tests to have good hearing. Dr. Hardy[2] found a somewhat higher average in a more extensive series of 50 ears. In comparison with the above is the prevailing notion that there are 14,000 fibres in the cochlear nerve. The clue to the original source of this determination was found in a footnote in an introductory book on physiological psychology,[3] where credit is given to McKendrick and Gray[4] (in Schäfer's Textbook on Physiology, 1900). McKendrick there states: "At my request, Dr. William Snodgrass made transverse section of the cochlear division of the auditory nerve of an adult who was not deaf and who did not die of a disease affecting the ear, and he carefully counted the number of nerve filaments. These were found to be about 14,000. It is obvious that all such estimations must be approximate." A later (1924) statement by Wilkinson and Gray[5] is to the effect that there are 20,000. A still later (1929) figure, given by Fletcher,[6] is 3,000. The original sources of these latter estimates have not been found.

MATERIAL AND METHODS.

Nerves were obtained during postmortem examination or later after the brain had been injected with formalin. The

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autopsies were performed from one to eight hours after death, except in two cases, where the elapsed time was 14 and 15 hours, but these bodies had been kept sufficiently cold to preserve the tissue satisfactorily. In all, 40 cochlear and 37 vestibular nerves were considered histologically normal, and all were from individuals with no history of hearing deficiencies, which, however, had not, in general, been checked by means of the audiometer, since many were cases of sudden or of accidental deaths. Among other causes of death were lobar pneumonia, lung abscess, peritonitis, septicemia, carcinoma of the bile duct, acute aplastic anemia, mitral stenosis, rheumatic endocarditis, uremia and brain tumor (not involving the ear or VIIIth cranial nerve). They were nearly all males, only five females being involved.

Because of the close association of the cochlear and vestibular fibres throughout most of their course, and the possibility of the cochlear portion of the nerve including at least saccular fibres,[7] it was assumed that the only way to get a reasonably accurate determination of the number of cochlear fibres would be to count the nerve fibres in the intrapontine portion of the vestibular nerve, central to the cochlear nuclei and distal to the point of bifurcation of the vestibular fibres, and deduct the resulting figure from the total number of fibres in the extramedullary portion of the whole VIIIth nerve. This procedure also seemed necessary because of the location of the ventral cochlear nucleus with reference to the vestibular nerve, the anatomical relations being such that some cochlear fibres might pass through the vestibular nerve (at an unknown distance peripheral to their attachment to the brain stem) in order to reach their destination in the rostral part of the cochlear nucleus. Accordingly, eight normal vestibular nerves were dissected out, as shown in Fig. 1.

Dissecting Procedure: After cleaning off the pia from the region of attachment of the cerebellar peduncles, removing the overhanging part of the cerebellum and removing the stalk of the flocculus, the Vth and VIIIth cranial nerves being carefully preserved, a curved probe is inserted between the tuberculum acusticum and the corpus restiforme, as shown in (A) of Fig. 1. The probe is worked peripherally between the cochlear and vestibular portions. The cochlear fibres, with much of the adherent cochlear nuclei, are reflected dorsally,

as in (B) of Fig. 1. By successive shallow incisions across the brachium pontis just lateral to the sensory root of the trigeminal nerve and lateral to the vestibular nerve, brachium pontine fibres are shredded off little by little, together

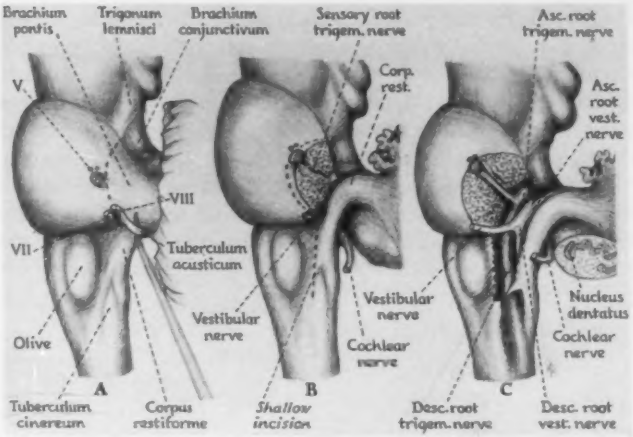

Fig. 1. Side view of adult human brain stem showing three stages in the dissection of the vestibular nerve and related structures.

Fig. 1A. The pia, most of the cranial nerves and ventral portions of the cerebellum (including the flocculus and its stalk) have been removed, exposing the cerebellar peduncles at their attachment to the brain stem. The probe is inserted between the cochlear nerve (with adherent cochlear nucleus) and the restiform body. The dotted line indicates the place of incision across the brachium pontis.

Fig. 1B. Cochlear portion of the VIIIth nerve has been separated from the vestibular and is reflected downward and posteriorly. Brachium pontis has been removed (layer by layer), with adherent portions of the cerebellum. The incision across the brachium pontis follows the lateral margin of the sensory root of the trigeminal nerve. The dotted line in the postolivary sulcus indicates the line of shallow incision (cutting ventral external arcuate fibres and dorsal spinocerebellar tract), which is necessary to displacement of the restiform body.

Fig. 1C. Restiform body has been displaced laterally, posteriorly and slightly downward, exposing the vestibular nerve with its ascending root (part of which disappears from view by passing medial to the brachium conjunctivum, and the rest passing lateral to this structure in company with the restiform body into the cerebellum) and descending root, which disappears beneath the restiform body. By carving deeper (to the dotted line across the brachium pontis of Fig. 1B), the trigeminal nerve, with its ascending and descending roots, may be brought into relief. For enumeration of fibres in the intrapontine portion of the vestibular nerve, sections were taken at the point where it crosses the descending root of the trigeminal nerve.

with such portions of the cerebellum as will naturally follow with the fibres. The incision is deepened, always remaining lateral to the penetrating fibres of the Vth nerve, until all of the brachium pontis has been removed. The specimen then

appears as in (B), Fig. 1. The superficial fibres (ventral external arcuate fibres and dorsal spinocerebellar tract) in the postolivary sulcus are then cut as far as the lower pole of the inferior olive. By placing a curved probe parallel to the ventral border of the restiform body and working it in between the restiform body and the vestibular nerve, it is possible to lift the restiform body from its bed and retract it dorsally so as to expose the vestibular nerve and much of its ascending and descending roots. The ascending root is a delicate ribbon that tends to adhere to the restiform body; but with care it can be separated off and followed upwards towards the brachium conjunctivum, where it splits into a portion that follows the restiform body (lateral to the brachium conjunctivum) into the cerebellum, and another portion that disappears underneath the brachium conjunctivum to end in the superior vestibular nucleus. The descending root of the vestibular nerve can also be followed downward under the restiform body and dorsal to the descending root of the trigeminal nerve almost to the nucleus gracilis and nucleus cuneatus. Just before the vestibular nerve divides into ascending and descending roots it crosses the descending part of the Vth nerve, as seen in (C) of Fig. 1. In this illustration the dissection has been carried farther in order to bring the trigeminal nerve into greater relief. The vestibular nerve is lifted from its bed by means of a smooth probe. Pieces of the vestibular nerve were taken from the region where it crosses the descending root of the Vth nerve. This dissection is rarely possible in fresh, unfixed brain stems, so that only two were considered satisfactory for staining with silver pyridine (after alcoholic fixation) and for osmic acid preparations; but in formalin-fixed brain stems, a high percentage of satisfactory dissections are possible. Formalin-fixed specimens are satisfactory for purposes of enumeration after being treated with Weigert's myelin sheath mordants for a couple of days, embedded in paraffin, cut at 6μ and stained by the Mallory-Heidenhain (azocarmine-orange G-aniline blue) method.

After comparing the fibre counts from the intrapontine portion of the vestibular nerve in silver, osmic acid and azocarmine preparations with that of the apparent vestibular portion (large-fibred region) of the corresponding whole VIIIth nerve, it was found unnecessary to make this dissection because a sufficiently close agreement existed between the

two vestibular counts. The accuracy of the fibre count in the intrapontine region is vitiated by the presence within the vestibular nerve (distal to the bifurcation) of an unsuspectedly large number of nerve cells, which are recognized as a vestibular nucleus (nucleus intraradicularis), giving origin to secondary vestibular fibres. This was not appreciated until longitudinal serial sections were made and these nerve cells counted. After these preliminary procedures on eight specimens, the remainder of the work was simplified by utilizing only cross-sections taken from the whole acoustic nerve some distance peripheral to its attachment to the brain. These were fixed in formalin, treated as listed above and stained by the Mallory-Heidenhain method. The region characterized by the presence of large fibres was regarded as vestibular and the rest as cochlear.

Counting Procedure: Enumeration was done by placing a Whipple ocular micrometer in one ocular of a binocular microscope and using an oil immersion lens. The micrometer squares the field and divides it into 100 smaller squares. By means of a smoothly working mechanical stage, whose axis of movement coincides with the lines in the micrometer, the section is explored systematically so that the entire surface is accurately and completely covered. Fractional fields are added to the whole fields and the number of fibres per individual field is recorded. Since there is enough histological difference between the cochlear division and the vestibular division, and the boundary between them rather sharp, cochlear and vestibular fibres can be recorded separately. A systematic procedure was followed with reference to including fibres whose axis cylinder touched two boundaries of the square and excluding those that touched the other two boundaries, as in blood counting.

Considerable effort was made to determine to what extent regularly placed samples of the cross-section area could be used instead of counting all the fibres. Several days of tedious work are required to carefully count all the fibres in a single nerve. By eliminating every other field and every other row of fields, or taking only every third field in every third row, etc., it was found sufficiently accurate to count only every third field in every third row or, roughly, one-ninth of the entire area. Such a count is frequently within 5 per cent of

the figure obtained by counting all the fibres. In this sampling method, the average number of fibres per field is obtained and then multiplied by the total number of fields represented by the particular component concerned. The time is thus reduced to half a day per nerve.

Incidentally, the dissection shown in Fig. 1 can be made a regular laboratory procedure in neuroanatomy. In (C) of this figure, the lower portion of the white lump, which automatically remains in the interior of the cerebellum as the fibres of the brachium pontis are removed, has been cut away to show that within is located the dentate nucleus. The medial surface of the vestibular nerve is smoother and demonstrates the division into ascending and descending roots better than does the lateral surface, which has been separated from the restiform body and may be a little more ragged. Since resection of the descending root (spinal tract) of the trigeminal nerve is becoming recognized as a surgical procedure for the relief of facial pain,[8, 9] its anatomical relations must be known more accurately than ever before. The dissection displays the cerebellar peduncles to maximum advantage.

RESULTS.

Funicular Pattern: The VIIIth nerve varies greatly in its cross-section appearance in different individuals and in different regions of the same nerve (see Fig. 2). At the bottom of the internal auditory meatus, both cochlear and vestibular divisions are represented by a large number of small bundles (see Fig. 2A), more or less separated from each other by ordinary fibrous connective tissue. These rapidly fuse, as is evident in Fig. 2B; which is about 2 mm. central to Fig. 2A. One or both components (see Fig. 2C) may remain throughout most of the remainder of the course as several fasciculi, the pattern of which may vary from level to level, due to funicular anastomoses. In a fair proportion of the nerves, two grossly distinct trunks are formed, but these rarely represent a true division into choclear and vestibular fibres, for in nearly all such cases there are at least a few obvious vestibular fibres in the cochlear trunk. In (D) of Fig. 2, nearly one-fifth of the apparent cochlear nerve is occupied by vestibular fibres. In surgical resection of what is presumably the vestibular nerve, about one-fourth of the vestibular fibres

in this case would escape being cut. The number of vestibular fibres thus remaining with the cochlear portion may be very

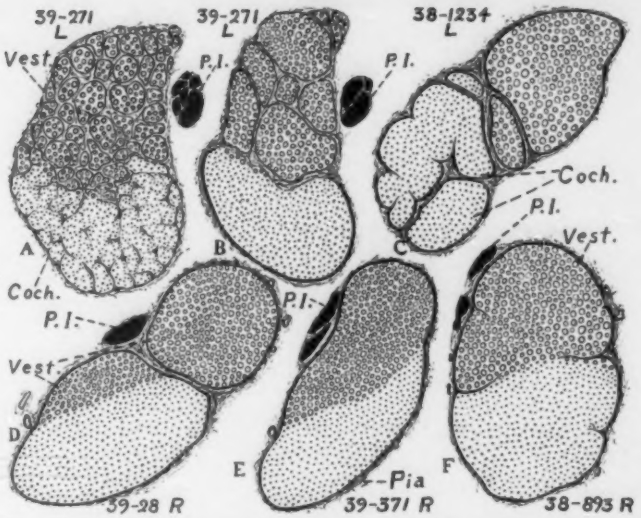

Fig. 2. A series of diagrams showing the funicular pattern of the VIIIth nerve from human adults. The cochlear and vestibular regions are distinguished from each other by different types of stippling. The solid black areas (P.I.) represent pars intermedia (nerve of Wrisburg) of the VIIth cranial nerve.

Fig. 2A. Cross-section slightly central to the vestibular ganglia. Both cochlear and vestibular portions consist of many small bundles, the cochlear having commenced to fuse.

Fig. 2B. A section of the same nerve as in Fig. 2A taken 2 mm. farther centrally, showing not only complete fusion of the cochlear but several vestibular fasciculi. The vestibular bundles ultimately fuse with each other and with the cochlear component until a single trunk is formed (similar to Fig. 2E). The heavy line about the lower part of the figure and limiting the fasciculi and whole nerve in Figs. 2C, 2D, 2E and 2F is the subpial membrane of glial tissue.

Fig. 2C. A case where neither cochlear nor vestibular completely fused through most of their course in the internal auditory meatus and subarachnoid space.

Fig. 2D. Example of the condition where the VIIIth nerve is represented by two distinct trunks, one of which was apparently purely vestibular, while the other was mostly cochlear, but with a prominent vestibular region amounting to about one-fifth of the cross-section area of the trunk.

Fig. 2E. Example of a common condition where there is a single trunk, a little more than half of which (upon microscopic examination) is found to be vestibular and the rest cochlear—the boundary between them being shown in Fig. 3.

Fig. 2F. A case where incomplete glial septa roughly indicate the division between cochlear and vestibular fibres. There are generally at least a few vestibular fibres on the cochlear side of such septa.

small. Fusion into a single trunk is common; but less common than gross appearance would suggest. Glial septa may be

present to more or less indicate the boundary, as in (F) of Fig. 2, or there may be no such demarcation, as is illustrated in (E) of Fig. 2, although sufficient magnification (of stained cross-sections) shows distinctly the limits of the two components (see Fig. 3).

Cross-Section Area: It is stated that the vestibular nerve is larger than the cochlear.[10] In most cases this was true, but in nine out of the 40 cochlear nerves the cochlear fibres occupied a distinctly larger area than did the corresponding vestibular. As a result, the averages of the entire series were the same, amounting to 144 micrometer fields, or 1.3 sq. mm. in the final histological preparation, which, of course, involves the usual shrinkage. The decrease in cross-section area due to the technique is assumed to be the same in the two divisions, although this assumption may not be valid.

Histological Structure: As is well known,[11, 12, 13, 14, 15] neuroglia tissue extends outward along the VIIIth nerve into the internal auditory meatus, and occasionally almost to the vestibular ganglion. It forms a prominent subpial membrane about each funiculus or trunk, and septal extensions may more or less subdivide the nerve. Glial strands or islands may be found as far peripherally as the vestibular ganglion. We have encountered a few small nests of nerve cells, as well as individual cells, at various distances outside the brain in the glial segment of the vestibular nerve, as reported by Tarlov.[15] This medial segment is not supported by connective tissue, except for a small amount which is associated with the blood vessels and a thin covering of pia. This part of the nerve is, therefore, fragile.

Distal to the glial extension, the nerve fibres possess a neurilemma and are supported by more connective tissue. With the technique used, the myelin sheath stained more intensely and appeared to be more dense in this distal (neurilemmal) region than in the glial segment. Due to the method of brain removal at routine autopsy, only part of the glial segment was available in many cases, hence all counts are based on this region.

Corpora amylacia were surprisingly common even in young children. When present, these globular bodies are associated with the glial tissue and stop abruptly where the glia ceases

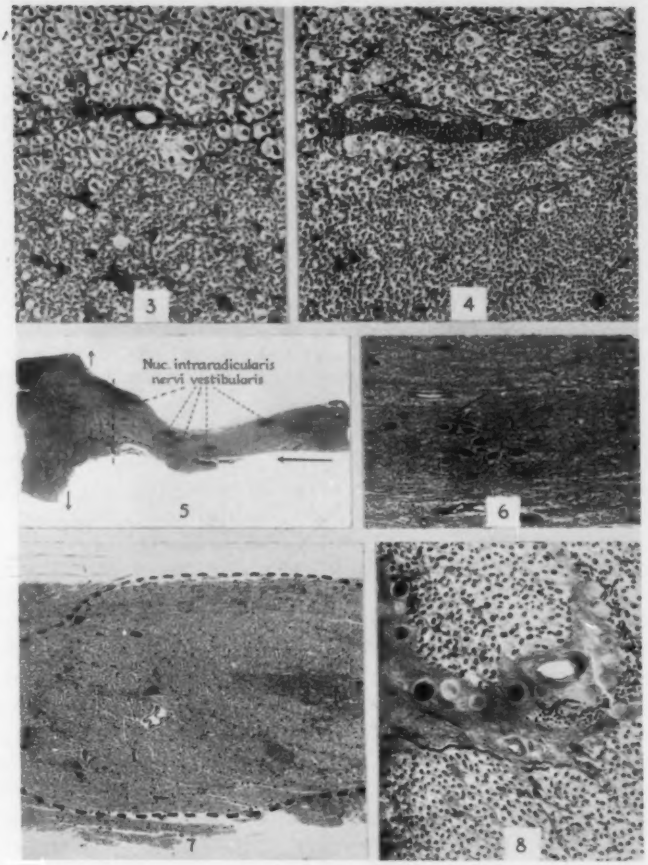

Fig. 3. Photomicrograph of the boundary between vestibular and cochlear portions of the extramedullary portion of the VIIIth cranial nerve, showing the difference in the histological appearance of the two components. Upper half, vestibular; lower half, cochlear. Formalin fixation. Weigert's mordants. Mallory-Heidenhain stain. 6 μ x 316. This and all other illustrations are from adult human specimens.

Fig. 4. Photomicrograph of the boundary between vestibular and cochlear portions of the extramedullary portion of the VIIIth cranial nerve, showing within the vestibular nerve an elongated area (dark in the picture) composed exclusively of very small (2-3 μ) fibres. There are about 700 fibres in the group shown. The extremely large vestibular fibres (mixed with scattered smaller ones) illustrate the other extreme encountered in the vestibular nerve. Upper two-thirds, vestibular; lower third, cochlear. Ammoniated alcohol. Pyridine silver. 5 μ x 250.

Fig. 5. Photomicrograph of a longitudinal section of the intrapontine portion of the vestibular nerve (dissected specimen) showing the location of several groups of cells (retouched) representing nucleus intraradicularis. The broken line indicates about the point where bifurcation begins. The ascending arrow, ascending root. Descending arrow, descending root. Horizontal arrow, main vestibular root. Same technique as Fig. 3. x 3-4/10.

Fig. 6. Photomicrograph of one of the groups of cells of nucleus intraradicularis nervi vestibularis shown in Fig. 5. x 134.

Fig. 7. Photomicrograph of a cross-section of the intrapontine portion of the vestibular nerve showing individual nerve cells scattered among the fibres. From the region where it crosses the descending root of the trigeminal nerve. Dissected specimen. The limits of the nerve are indicated by heavy broken line. Same technique as in Fig. 3. x 134.

Fig. 8. Photomicrograph of the margin of the VIIIth nerve in the boundary between vestibular and cochlear portions showing a few unmyelinated fibres (running horizontally in the section) entering with the vascular stroma. The round light and dark areas in the glial septum are corpora amylacea. Many fine fibres are present in the vestibular division adjacent to the trabecula. Upper half, vestibular; lower half, cochlear. Same technique as in Fig. 4. x 316.

and neurilemma commences. Even when abundant in the VIIIth nerve, they are absent in the adjacent pars intermedia of the VIIth nerve, which is supported by connective tissue rather than neuroglia.

Even moderate magnification shows a distinct difference between the cochlear and vestibular regions. The cochlear fibres are more uniform in size and appear more compact. The vestibular portion, on the other hand, presents a more open appearance, due to the presence of a great many very large fibres, as in the upper half of Fig. 3.

The vast majority of the cochlear fibres are 5 μ to 7 μ, although they range from 3μ to 10 μ. The vestibular fibres vary from 2 μ to 15 μ; most of them are at least 10 μ. Many of the small vestibular fibres are frequently concentrated in limited regions and usually near the margin of the nerve. A rather extreme case is shown in Fig. 4, where about 700 very small vestibular fibres, unmixed with large ones, occupy an elongated region extending across the entire figure and are surrounded by very large vestibular fibres. This group is near the junction with the cochlear components. According to Lorente do Nó,[16] these small fibres are mostly connected with the periphery of the cristae of the semicircular canals, and restricted regions of the macula sacculi.

The average relative size is probably best appreciated from the fact that, with the equipment used, the oil immersion field embraced by the ocular micrometer (95 x 95 μ, or 9,000 sq. μ, in round numbers) contains an average of 224 cochlear fibres and only 131 vestibular fibres. The variations in diameter of the fibres correlate well with the differences in the size of the nerve cells of the spiral and vestibular ganglia, since Alexander[17] found the spiral ganglion cells of man to measure

from 13 μ to 16 μ, whereas the vestibular ganglion cells are from 19 μ to 27 μ.

A few unmyelinated fibres are regularly encountered. These are usually associated with the vascular stroma, as illustrated in Fig. 8, which is taken from the periphery of the nerve in the boundary zone between cochlear and vestibular fibres where some blood vessels are entering. Because of the close association of these unmyelinated fibres with the blood vessels, they are probably vasomotor in function. Fig. 8 also shows a concentration of fine fibres in the vestibular portion (upper half) adjacent to the trabecula.

Additional details may be found in a review of the applied anatomy of the VIIIth cranial nerve by Courville.[18] For the anatomy of the spiral ganglion, see Wolff,[19] and on the distal processes of these ganglion cells and their relation to the organ of Corti, the reader is referred to the work of Poljak[20] and Lorente do Nó.[21]

The Intrapontine Portion of the Vestibular Nerve: After entering the pons, the vestibular nerve is more or less ovoid in cross-section, one diameter being two to three times the other. It is subdivided by glial septae into numerous inconspicuous oblong and spindle-shaped fasciculi, as illustrated in Fig. 7. At various points distal to the division into ascending and descending roots, groups of multipolar nerve cells, as shown in Figs. 5-7, and individual cells, as in Fig. 7, are encountered among the vestibular root fibres. Fig. 5 is a low power view of a longitudinal section of the intrapontine portion of a dissected vestibular nerve, including part of its ascending and descending roots, showing the location of six groups of cells. One of these groups is shown at a higher magnification in Fig. 6. These nerve cells vary greatly in size, as is shown best in Fig. 7. They tend to be elongated in the direction of the fibres. Collectively, they represent what Klossowsky[22] called the nucleus intraradicularis nervi vestibularis, which is considered homologous to the nucleus vestibularis tangentialis of birds, reptiles and teleosts.[23, 24] Since it is recognized as one of the terminal vestibular nuclei, reasonable accuracy in the determination of the number of vestibular fibres in this portion of its course cannot be assured if these cells are very abundant, because an unknown number

of primary vestibular fibres would have terminated about the cells distal to the point of enumeration, and the primary vestibular fibres would be mixed with a variable number of fibres of the second order. On the assumption that the number of nerve cells in the intraradicular nucleus was relatively small, counts were made on eight roots at the point where the vestibular nerve crosses the descending trigeminal root (see Fig. 1). The number varied from 14,700 to 22,600, with an average of 20,100. The large-fibred portion of the VIIIth nerve of the same individuals before entering the pons contained from 15,500 to 23,600 fibres, with an average of 20,700. Although the averages happen to be essentially the same, the count in the two regions of the same nerve may vary by nearly 3,000 fibres. At times, the larger number was intrapontine, and at other times extrapontine. In view of this situation, it appeared just as accurate to confine the enumeration to the fibres in that portion of the acoustic nerve which is outside of the brain, and to separate the vestibular fibres from the cochlear fibres on the basis of the histological appearance, as already described. All the data in Table I were obtained in this manner.

TABLE I.

Number of Nerves	Age Yrs.	Range	Vestibular Portion	Cochlear Portion	Total VIIIth Nerve
19 Vest.	2	Min.	15,300	26,100	43,800
and	to	Max.	24,000	40,000	62,600
21 Coch.	26	Aver.	18,900	32,500	51,700
18 Vest.	44	Min.	14,200	22,800	38,300
and	to	Max.	22,900	38,500	61,400
19 Coch.	60	Aver.	18,000	30,300	48,000
37 Vest.	2	Min.	14,200	22,800	38,300
and	to	Max.	24,000	40,000	62,600
40 Coch.	60	Aver.	18,500	31,400	49,900
Maximum difference between right and left			4,000	4,700	8,600

The number of nerve fibres in the VIIIth cranial nerve of man, based on 37 normal vestibular and 40 normal cochlear nerves, showing the lower limit, upper limit, average and greatest difference between the right and left sides of the same individual.

It still seemed desirable to obtain some definite idea as to the number of nerve cells found among the fibres of the main vestibular root in its course within the pons and distal to the better known vestibular nuclei. For this purpose, three dis-

sected vestibular nerves were compressed between glass slides during part of the time while being fixed in formalin as a means of straightening the curves. They were cut serially at 6 μ in the longitudinal direction (see Fig. 5) and stained by the Mallory-Heidenhain method. The number of nerve cells containing nucleoli in every tenth section distal to the broken line of Fig. 5 was determined. As many as 66 cells were encountered in a single section, with an average of 29 per section for the 17 sections used, with a total of nearly 5,000 cells in the entire series, which in this case consisted of 200 sections. In another specimen, there were 3,900 cells, and in a third only about 2,300 cells. Obviously such a large number of cells makes it impossible to determine accurately the number of primary vestibular fibres from the intrapontine segment. In addition to this drawback, there are several other sources of error, such as the complex arrangement of the fibres, indefinite nerve boundaries in some cases, and the possibility that not all of the root has been retained in the dissection. In Fig. 5, where a broken line has been drawn around the limits of the vestibular fibres, it is seen that a little of the root might have been dissected away at the upper centre region and that some adherent tissue has been retained at the upper left corner of the illustration, as well as at the lower margin.

The Number of Cochlear and Vestibular Fibres: The data on 37 vestibular and 40 cochlear nerves are tabulated in Table I. More vestibular nerves than cochlear contained pathological features which automatically ruled them out. This accounts for the unequal number of specimens. Since there was a large gap in the series with reference to age, those below age 27 years have been summarized for comparison with those between age 44 and 60 years. Such a division would be desirable even if the series were continuous since Bunch[25] and others have noted a decrease in auditory acuity, especially for high tones, after age 30 years. Unfortunately, the cases here involved were not in general tested with the audiometer; but there is no reason for assuming that the usual age changes do not apply to this material.

The number of vestibular fibres within the whole group varied from 14,200 to 24,000, and averaged 18,500. The last two or three digits are not significant, hence all figures are

more or less in "round" numbers. The error is undoubtedly greater in the vestibular figures than in those pertaining to the cochlear because of the number and irregular distribution of very small fibres in the vestibular division. There are no comparable fibre or cell counts, but from the inherent sources of error one would expect the number of vestibular fibres to be somewhat higher had all the fibres in silver preparations been counted. The error in the average figure probably does not exceed 1,200 fibres, but may be as high as 2,000 in a few individual cases. It would require much more work than the results would justify to establish this point with statistical accuracy. Since the factors responsible for these errors are operating in the same direction in both age groups, marked differences in the averages may be assumed to be real.

There are nearly 1,000 fewer vestibular fibres in the older group. Of the apparently equally normal ears of one individual, one may have 4,000 fewer vestibular fibres than the other. This is not an unsuspected range of variability, considering biological material in general and, more specifically, the correlation of morphological data and acuity of hearing brought out by Guild, Crowe and co-workers at the Otological Research Laboratory, Johns Hopkins University, during the past 10 years.

Apparently normal cochlear nerves contain from 22,800 to 40,000 nerve fibres, the average being 31,400, which are distinctly higher figures than any fibre counts on human material thus far discovered in the literature. The close agreement between these data and the independent figures on the number of spiral ganglion cells[1] makes it safe to regard the number of conductors carrying auditory impulses into the human brain under normal conditions as being anywhere from about 23,000 to 40,000. The average number of cochlear fibres in man appears to be between 31,000 and 32,000, as compared with nearly 50,000 in the cat (Howe[26]).

The older group contained 2,200 fewer cochlear fibres than the younger group. One might expect even a greater difference than this. Still older people might show much greater loss; but our purpose was to select nerves from individuals with hearing reasonably close to normal. We have purposely avoided very old specimens.

Again, the right and left nerves of the same individual may vary by as much as nearly 5,000 cochlear fibres, without ordinary tests showing hearing disability. Corresponding data would be highly desirable on audiometer-tested cases. In general, however, if the number of cochlear fibres of a given individual are distinctly below the average on one side, they are also below the average on the other side, and if well above the average on one side, those on the other are usually also above the average. The same is true of vestibular fibres.

SUMMARY.

A histological study of 37 normal vestibular nerves and 40 normal cochlear nerves from individuals ranging from age 2 to 60 years shows:

1. The funicular pattern of the VIIIth cranial nerve of man varies greatly in different individuals, and in different regions of the same nerve. When two distinct trunks are present, one is probably wholly vestibular, but the other is cochlear, with a variable number of vestibular fibres.

2. The pars intermedia of the VIIth nerve is usually closely associated with the pial covering of the vestibular portion of the VIIIth, and hence is frequently retained with the vestibular component without any special attempt to include pars intermedia in the section.

3. The cross-section area of the vestibular portion is usually the larger, but in about one-fifth of the cases the cochlear was distinctly the larger. The average cross-section area of the entire series was the same for the two components.

4. Only a few unmyelinated nerve fibres are present, and these are usually in the neighborhood of blood vessels.

5. A method of dissecting the vestibular nerve with its ascending and descending branches within the pons and medulla oblongata is described and illustrated.

6. The number of nerve fibres in the intrapontine portion of the main vestibular nerve agrees sufficiently with the number of nerve fibres in the large-fibred portion of the extrapontine portion of the VIIIth nerve to indicate that, for practical purposes, the more uniform and smaller-fibred portion may be regarded as cochlear.

7. Among the fibres of the vestibular nerve within the pons and distal to the origin of the ascending and descending vestibular roots may be as many as 5,000 nerve cells which represent the nucleus intraradicularis nervi vestibularis. This fact mitigates against the accuracy of any determination of the number of primary vestibular fibres based on the intrapontine region of the vestibular nerve.

8. If the region of the extramedullary VIIIth nerve that is characterized by the presence of large fibres be regarded as the vestibular component, then the vestibular fibres vary from 14,000 to 24,000, with an average of 18,500. On account of the irregular distribution of very small fibres among the larger ones, these figures are probably somewhat too low, due to the sampling method used.

9. On the assumption made in No. 6 above, cochlear fibres vary from 24,000 to 40,000, with an average of over 31,000. These figures agree with the number of spiral ganglion cells and may, therefore, be regarded as the number of auditory conductors entering the brain of normal individuals.

10. The vestibular and cochlear components in a particular individual tend to vary from the average in the same direction, and the right side tends to vary directly with the left side; but there are exceptions. The right may vary from the left (of the same individual) by as many as 4,000 vestibular fibres, 5,000 cochlear fibres and 8,500 fibres in the entire VIIIth nerve.

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NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLARYNGOLOGY.

Meeting of May 3, 1939.

(Continued from December, 1939, issue.)

Carcinoma of the Nasopharynx with Extension to the Petrous Pyramid. Dr. Joseph G. Druss.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. A. A. EGGSTON: This is a very interesting case. I don't know where Dr. Druss thought the primary lesion was. This is quite typical of a so-called Schmincke tumor — that is, one which infiltrates the mucosa and submucosa. In the section of the petrous bone, I didn't get whether the tumor had infiltrated all the petrous pyramid or not. These are very interesting tumors and special attention should be drawn to them, because very frequently the primary lesion is overlooked until metastasis has occurred.

DR. JOSEPH G. DRUSS: As far as Dr. Eggston's question with regard to the extent of involvement in the petrous pyramid is concerned, I wish to state that only the apex was infiltrated by the tumor mass. The carcinoma extended from the nasopharynx to the basisphenoid and to the apex of the petrosa. It did not reach as far laterally as the perilabyrinthine region, although secondary inflammatory reaction at this site was present. Emphasis was placed on the difficult problem in diagnosis, which was made in this case relatively late in the disease.

(a) — Needle in Aorta.

(b) — Bilateral Empyema (Leptothrix). Dr. David H. Jones.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. FRANCIS W. WHITE: I didn't see the first case Dr. Jones has presented, but I followed the second case all the way along, and it was one of the saddest I have ever seen, watching that child go downhill day by day.

An effort has been made to have legislation passed prohibiting the sale of these dangerous corrosives, but I don't believe it has gotten very far as yet.

Experimental Evidence of Gonadotropic Hormone in Nasal and Sinus Mucous Membranes. Dr. A. A. Eggston.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

The Genitonasal and Genitoaural Relationships. Dr. Hector Mortimer.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. SAMUEL ROSEN: I would like to bring out just one point: the work of both Dr. Mortimer and Dr. Eggston has concerned itself primarily with hormonal effects on the nasal mucosa or of the nasal mucosa on the sexual

organs. For a few years, we also have been doing experimental work in this field at the Mt. Sinai Hospital in New York. The thing that fascinated me was the neurological aspect of the nasogenital relationship.

We have taken a strong solution of silver nitrate, painted the nasal mucosa of the rat, and have been able to produce pseudopregnancy. We did not know at that time whether this phenomenon was due to the stimulation or irritation of the nerve-endings in the nose. We, therefore, used nupercaine and blocked out the sensory nerves of the nose. It was very interesting to note that we obtained the same result (pseudopregnancy) with the nupercaine that we had with the silver nitrate, so it is probable that the latter, by causing an eschar temporarily at least destroyed the sensory perception of those nerves. We devised an operation of sphenopalatine ganglionectomy in the rat. It was interesting to find that again we obtained those same changes in the sexual organs after ganglionectomy — true pseudopregnancy with decidual changes in the uterus.

At the present time we are carrying our studies further. As a hypothesis, we believe there is present a nervous pathway from the nose to the pituitary.

DR. LEE HURD: I have tried this treatment on four patients with otosclerosis, one of whom improved under treatment. Patients having the nerve type of deafness, however, do not respond — simply those having otosclerosis.

DR. FRANCIS W. WHITE: I am sure we all appreciate this wonderful demonstration by Dr. Mortimer. We thank him for coming here and clarifying certain phases of his work. It is to be hoped many may follow his suggestions, so that in a short time comprehensive clinical reports may be at hand.

FOURTH INTERNATIONAL OTO-RHINO-LARYNGOLOGICAL CONGRESS.

The Committee of the Fourth International Oto-Rhino-Laryngological Congress regret that they are obliged to postpone the Congress for an indefinite time.

They hope that the present horror will again give way to better times and that at some future date they may be permitted to send their invitation to the oto-rhino-laryngological world. H. Burger, President; A. Marres, Honorary Secretary.

BOOK REVIEWS.

Tuberculosis of the Upper Respiratory Tracts. By F. C. Ormerod, M.D. (Manch.), F.R.C.S., Eng. Surgeon to the Throat and Ear Department, Brompton Chest Hospital. Consulting Laryngologist, Royal National Hospital for Consumption, Ventnor. Surgeon, Golden Square Throat, Nose and Ear Hospital. Assistant Surgeon, Ear, Nose and Throat Department, Westminster Hospital. Two hundred fifteen pages with Index and 55 illustrations, 31 plates of which are in color. London, W.: John Bale, Medical Publications, Ltd., 85 Great Titchfield street. 1939. Price 21/net.

The book is divided into four sections as follows: Section I deals with tuberculosis of the larynx; Section II with tuberculosis of the pharynx and its associated structures; Section III with tuberculosis of the nose, accessory sinuses and nasopharynx; and Section IV with tuberculosis of the ear.

Each section begins with an historical note. The author discusses the incidence, etiology, pathology, frequency in different parts of the larynx, clinical features, symptoms, differential diagnosis, general considerations of treatment of laryngeal tuberculosis, galvanocautery and surgical treatment, X-ray and light therapy, relief of pain, prognosis, end-results, tonsillectomy and lupus, all under the first section. There are seven photomicrographs, 18 colored illustrations, five photographs of gross specimens, three in gray of tuberculosis of the larynx and six tomograms. The tomograms show the asymmetry of the larynx caused by a tuberculous lesion and the location of the lesion.

Under Section II he presents the diseases of the pharynx, trachea, bronchi, esophagus and tonsils. One colored illustration shows an acute miliary infection of the uvula, pillars and left fauces. Another shows an extensive ulceration of the posterior wall of the oropharynx. A third colored illustration of a postmortem specimen shows a large ulcer over the posterior wall of the hypopharynx. A fourth in colors shows extensive ulceration of both tonsils. Ulceration of the lateral margin of the tongue is well illustrated in colors in a fifth. The sixth demonstrates an ulcer of the floor of the mouth, angle of the lips and alveolar margin. The illustrations in color really show what the author saw in his patients' lesions. In no other way can tuberculous lesions be so well produced.

Three large, full page illustrations, two of which are in colors and the third in gray, show autopsy specimens of early ulceration of the trachea and bronchi. One of these demonstrates the importance of a tuberculous lymph node in occluding the lumen of the trachea. This last point has been emphasized by several bronchoscopists in this country during the past few years, and especially by Kernan and Myerson.

There is one excellent colored illustration of tuberculosis of the esophagus showing ulceration of the upper third.

There are four colored illustrations of lupus and tuberculosis of the nose, and three illustrations in color of aural tuberculosis, with five others, in gray, of the drum.

The author is to be commended for presenting tuberculosis of all the upper respiratory tracts, including the ear, and for including the lupus, which is so often omitted except by writers on the Continent. The text is well arranged and the entire subject is very logically presented in detail.

The photographs, micrographs and colored illustrations are all well done. These amplify the text and emphasize the important points in the book. The bibliography consists of 313 refcrences. F. R. S.

Medicine of the Ear. By Edmund Prince Fowler, Jr., M.D., Sc.D. Editor. Assistant Clinical Professor of Otolaryngology, College of Physicians and Surgeons, Columbia University; Assistant Surgeon, Manhattan Eye, Ear and Throat Hospital, New York. With foreword by John Devereux Kernan, M.D., Professor of Otolaryngology, College of Physicians and Surgeons, Columbia University. Six hundred three pages with Index. New York: Thomas Nelson and Sons, 1939.

This book is published as a companion book to Kopetzky's Loose-Leaf Surgery of the Ear and consists of 17 monographs, each dealing with a separate division of nonsurgical otology. Each chapter represents an authoritative exposition of the most modern conceptions of a special phase of otology and its relationship to general medicine.

No textbook in the last decade has brought such important contributions from the physicist and the chemist to the diagnosis and treatment of ear diseases, and this book has correlated all of this important information and presents it in usable form to the otologic clinician. Of necessity, certain sections are more or less elementary and easily understandable even to the beginner in medicine and to the layman interested in the subject; but certain other parts are technical enough to satisfy the demands of even the most erudite of research investigators.

Out of the wealth of modern literature dealing with ear diseases, both from the standpoint of the pathological mechanisms involved and their causation, and the most modern methods available for their diagnoses and medical treatment, these authors have taken the best and presented it in a manner which is both comprehensive and useful.

This book is sincerely recommended to the thinking otologist. A. M. A.